“A RETROSPECTIVE STUDY OF DEATHS DUE TO POISONING, AMONG THE AUTOPSIES CONDUCTED AT GOVERNMENT KILPAUK MEDICAL COLLEGE & HOSPITAL, CHENNAI”

Dissertation submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfillment of the requirements

For the award of degree of

M.D. (FORENSIC MEDICINE)

(Branch-XIV)

DEPARTMENT OF FORENSIC MEDICINE

GOVT. KILPAUK MEDICAL COLLEGE

CHENNAI - 600010.

THE TAMILNADU DR.MGR MEDICAL UNIVERSITY

CHENNAI-600032

APRIL-2017

CERTIFICATE

This is to certify that this dissertation titled **"A RETROSPECTIVE STUDY OF DEATHS DUE TO POISONING, AMONG THE AUTOPSIES CONDUCTED AT GOVERNMENT KILPAUK MEDICAL COLLEGE & HOSPITAL, CHENNAI"** bonafied original work done by **Dr. S. SYLVIA** Post graduate in Department of Forensic Medicine Govt. kilpauk medical college Chennai, in partial fulfillment of the regulations of the Tamilnadu Dr.MGR University for the award of M.D. Degree in Forensic medicine (Branch XIV)

Dr. R. NARAYANA BABU M.D., DCH.,
DEAN
Government Kilpauk Medical College and Hospital, Chennai – 600 010.

Dr. R. SELVAKUMAR M.D.,
Professor and HOD,
Department of Forensic Medicine,
Government Kilpauk Medical College and Hospital,
Chennai – 600 010.

CERTIFICATE

This is to certify that this dissertation titled **"A RETROSPECTIVE STUDY OF DEATHS DUE TO POISONING, AMONG THE AUTOPSIES CONDUCTED AT GOVERNMENT KILPAUK MEDICAL COLLEGE & HOSPITAL, CHENNAI"** submitted by **Dr. SYLVIA.S** is an original work done in the Department of Forensic Medicine, Government Kilpauk Medical College and hospital , Chennai in partial fulfillment of regulations of The Tamil Nadu Dr. M.G.R. Medical University, for the award of degree of M.D. (FORENSIC MEDICINE) Branch – XIV, under my supervision during the academic period 2014-2017.

Dr. R. SELVAKUMAR M.D.,
Professor and HOD,
Department of Forensic Medicine,
Government Kilpauk Medical College and Hospital,
Chennai – 600 010.

DECLARATION

I, **Dr. SYLVIA. S** solemnly declare that the dissertation on **"A RETROSPECTIVE STUDY OF DEATHS DUE TO POISONING, AMONG THE AUTOPSIES CONDUCTED AT GOVERNMENT KILPAUK MEDICAL COLLEGE & HOSPITAL, CHENNAI"** is a bona-fide work done by me during the period of August 2015 to August 2016 at Government Kilpauk Medical College and Hospital, under the expert Supervision of **Dr. R. SELVAKUMAR, M.D,** Professor and Head of Department of Forensic Medicine, Government Kilpauk Medical College, Chennai. This thesis is submitted to The Tamil Nadu Dr .M.G.R. Medical University towards partial fulfillment of the rules and regulations for the M.D. degree examinations in Forensic Medicine to be held in April 2017.

Dr. SYLVIA.S.

Place: Chennai-10
Date: 28/ 09/ 2017

ACKNOWLEDGEMENT

I wish to thank **Dr. R. NARAYANA BABU MD., DCH**, Dean, Govt. Kilpauk Medical College and Hospital, Chennai.

With sincere gratitude and I wish to acknowledge the expert guidance and suggestions of my Professor and HOD, **Dr. R. SELVAKUMAR, MD.,** without whose permission and guidance this study would not have been possible.

I am deeply indebted and grateful to my co-guide **Dr. G. Manigandaraj MD.,** Asst. Professor, Department of Forensic Medicine**,** Govt. Kilpauk Medical College and Hospital, Chennai for his valuable support and guidance, I wish to thank **Dr. I. Jithender Singh and Dr. S. Devasena,** Department of Forensic Medicine, Govt. Kilpauk Medical College, Chennai for their able guidance, constant inspiration and continuous encouragement rendered at every stage of this study.

I am deeply indebted to and highly grateful to **Dr. K.VINOTH M.D., and Dr. S. SHANKAR M.D.,** Department of Forensic Medicine, Govt. Kilpauk Medical College, without whom this work would not be in the present shape. I wish to thank all my co-post graduates for helping me in this work. I also wish to thank all the non-teaching staffs in my department for helping me throughout the study.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

Poison is a substance which produces toxicity. The term Poison connotes a high probability of the toxic effects produced by a substance[1.] Poison can be defined as any substance (solid, liquid or gas) which if introduced into a living body or brought in contact with any part will produced ill health or death by its constitutional or local effects or both[2.] Paracelsus the father of toxicology (1493-1541) wrote

"All things are poisons and there is nothing that is harmless, the dose alone decides that something is no poison" [3.] Drugs are substance used for the treatment of disease, which produce a beneficial effect with minimum bad effect[1.]

It has been calculated that some form of poison directly or indirectly was responsible for more than 1 million illness world-wide annually, and this calculation could be just the tip of iceberg since most cases of poisoning actually go unreported. Every year in India it is estimated that more than 50,000 people die from toxic exposure[4.]

Poison consumption as a mode of death is known from ancient times. Poisoning is seen among all age groups and both sexes everywhere. The incidence of poisoning with reference to insecticides, cleaning acids, pesticides and hair dye has become more common than the others in the recent times. It is because of increased availability and indiscriminate use of the various pesticides in agricultural areas[5.] The trend in poisoning show a change due to introduction of newer pesticides under different classes. At one point historically arsenic was the most popular besides Copper Sulphate, and Barbiturates. In the recent past DDT, Benzene Hexa Chloride, Endrin, Organo-

Chlorines and Organo-Phosphorous compounds took the toll. Of late Aluminum Phosphide, Alcohol, hair dye and Carbamates are in vogue[5.]

All poisoning death cases are recorded as unnatural death. A medico-legal autopsy is routine. This is according to the legal system of our country. In the present century poisoning as become a major epidemic of non-communicable disease. Suicide, a major problem posed earlier amongst the industrialized nations has shifted its paradigm into the developing countries. Deaths due to poisoning come next to road traffic accident death, among the unnatural deaths according to National crime record bureau India[6.] Poisoning can be accidental exposure or intentional exposure. Many studies have shown that mortality is high in deliberate self-poisoning.

Pattern of poison is influenced by different factors like, occupation, socio economic status and emotional impact. Pattern of poisoning varies in every place. Knowing the pattern of poisoning cases in a region helps in suggesting proper earliest preventive measures and also in early management of cases.

This study will aim at determining socio demographic profile, pattern of poisoning during the study period. Therefore the findings of this study will be helpful for the government authorities and planning bodies, to plan and implement strategies towards prevention of poisoning. The rising incidences of Suicide poisoning with prevalence, made us to undertake this study to know the patterns, trends & other factors of deaths due to poisoning and thereby drawing attention of the health policy makers to enhance the legislative measures and prevent such casualties.

AIM AND OBJECTIVES

1. To study the trends in death due to poisoning.
2. To profile the factors involved in poisoning.
3. To study the manner of poisoning.
4. To identify the factors contributing to poisoning
5. To study the Post mortem finding in poisoning cases.
6. To identify the most common poison causing deaths in suicidal cases and accidental cases.
7. To compare the history, post mortem finding and forensic science lab results.
8. To suggest preventive measures, that can be adopted to prevent poisoning.

REVIEW OF LITERATURE

Legal aspects of Poisoning:

All the hospital (Government run or privately owned) is under a law to treat a case of poisoning.[4]

All homicidal poisoning cases must be reported to the police as per section 39 CrPC.and failure to report, will make them culpable under section 176 IPC.[4]

- 202 and 193 IPC – if the doctor withholds or gives wrong information aboot the case, he is punishable under this.
- 175 IPC- any information required by the police should be disclosed, no role of professional secrecy.

Doctors should collect relevant sample and send it to forensic science laboratory. If the patient is about to die, dying declaration can be arranged accordingly. If the patient dies before diagnosis, information to be reported to police and sent for medico legal autopsy[4]

Consent

A patient, who has deliberately consumed a poisonous substance or overdosed on a therapeutic drug, is likely to be uncooperative and may resist all efforts at treating him. The attending physician may then be uncertain as to the legal implications of forcing treatment on the patient who may even threaten the doctor with a law suit if therapeutic procedures are forcefully carried out.[4]

It is however a fact that a patient who attempts suicide **has lost the right to refuse treatment**, and no court has so far upheld a patient's complaint of "forced treatment" in such circumstances. It is also true that in many such cases of toxic ingestions, the patient can be declared "**not rational enough to refuse treatment"** on account of depression or disturbance of mental functions which can be deemed to impair judgement. On the other hand, a physician may become liable for negligence if he does not do what is medically indicated.[4]

Legal provisions

1. **Sec – 176 IPC:**

   Provides punishment for omission to give notice or information (including that of a poisoning case) to the public servant/police.[7]

2. **Sec - 177 IPC:**

   Provides punishment for furnishing false information (including that of a poison case).[7]

3. **Sec – 201 IPC:**

   Provides punishment for causing disappearance of evidence of offence (e.g. destroying sample of gastric lavage, clothing carrying evidence of poison etc.)[7]

4. **Sec - 202 IPC:**

   Provides punishment for intentional concealment of information of offense (including that of a poison case).[7]

5. **Sec - 269 IPC:**

   Negligent act which is likely to spread infection which is dangerous to life.[7]

6. **Sec – 270 IPC:**

   This is Malignant act - spread infection of disease which is dangerous to life.[7]

7. **Sec - 272 IPC:**

   Act is about adulterating of the food or the drink which is intended for sale.[7]

8. **Sec – 273 IPC:**

   Act is about selling of noxious food or drink.[7]

9. **Sec – 274 IPC:**

   Act is about adulterating of drugs.[7]

10. **Sec – 275 IPC:**

    Act is about selling of adulterated drugs.[7]

11. **Sec – 276 IPC;**

    Act is about selling of a drug as a different drug or different preparation.[7]

12. **Sec – 277 IPC:**

    Act is about polluting water of public spring or reservoir.[7]

13. **Sec – 278 IPC:**

    Act is about making atmosphere noxious to health.[7]

14. Sec – 284 IPC:

Prescribes for rash or negligent act (or omission) in relation with poisonous substance so as to endanger human life or to be likely to cause hurt or injury to any person.[7]

15. Sec – 299 IPC:

Culpable homicide including that caused through administration of poisonous substance.[7]

16. Sec – 300 IPC:

Murder including that caused through administration of poisonous substance with the intention of causing death.[7]

17. Sec – 304 – A IPC:

It is death by negligent / rash act (or omission) including that caused through poisoning.[7]

18. SEC-304B – IPC:

Dowry death.[7]

19. SEC- 320- IPC:

Grievous injury.[7]

20. Sec – 324 IPC:

Makes simple hurt more grave and liable to a more severe punishment – where it has been inflicted by one of the means. Poison or any substance that is deleterious to the human body to inhale, swallow, or to receive into the blood.[7]

21. Sec – 326 IPC:

It is similar to Sec – 324 with the only difference that the words "grievous hurt" has been substituted for the word 'hurt' providing enhanced punishment.[7]

HISTORICAL ASPECTS OF POISONING

The history of poisons and poisoning dates back to thousands of years. Since then poisons have played an important role in human history ranging from individual poisoning to political-assassinations and to environmental concerns.[4]

Poisons of antiquity

The Ebers Papyrus, an ancient Egyptian text written about 1500 B.C. and is considered as earliest medical records, describes many ancient poisons, including Arsenic, Antimony, Lead, Opium, Mandrake, Hemlock, Aconite, Wormwood & Glycosides.[4] The earliest poisons consisted of plant extracts, animal venom & minerals. These poisons are thought to have mystical properties and their use was surrounded by repetition and intrigue.Much literary work has reference of arrow poison. The ancient Indian book Rig Veda – (12^{th} century B.C) refers to the use of Aconitum species for arrow poisons. In Odyssey, Homer (850 B.C.) wrote about the use of a variety of poisons in his arrows.

During Greek and Roman times, the firm attempts were taken at poison identification, classification and introduction of Antidotes.Categorization of

poisons was first done by Dioscorides (A.D. 40-80) in *Materia Medica* by their origin: animal, vegetable or mineral.Animal poison usually referred to is the venom from poisonous animals, including snake, toads, salamanders, jelly fish and stingrays. Nicander (204 – 135 BC), presented the manifestation and treatment of the effect of poisoning by means of the poem "*Theriaca*". Vegetable poisons were described by Theophrastus (370 – 280 BC) in his treatise "*De Historia Plantarum*" which includes vegetable poisons as Aconite, Hellebore, Henbane, Mandrake, Hemlock etc.

Aconite was described as "**Queen mother of poisons**"

Hemlock was described as the official **state poison** used by the Greeks and was employed in the execution of Socrates (470 – 399 B.C.).

Mineral poison like Lead, was known as early as 3500 B.C., had a role in the fall of the Roman Empire.

The chlorinated hydrocarbon- DDT was synthesized first in the 19th Century, but its insecticide properties were not discovered until 1939. It was used during the Second World War for the control of insects. From 1961 onward its use has become less and was finally withdrawn. However, in India DDT is still available and used.

Aluminum Phosphide has become a more common poison and it is being used as a pesticide since 1940. Over the last decade poisoning due to Aluminum

Phosphide poisoning has been reported widely from different Northern states of India and its incidence is progressively increasing in the northern India.[4]

Important figures in toxicology during medieval and renaissance time Scientists:

Maimonides (1135-1204 B.C.) – Treatise on Poisons and their Antidotes in 1198 B.C.[3] Paracelsus (1493 – 1541 B.C.) – Study on dose response relationship – beginning of the scientific approach to toxicology. He stated "*What is there that is not poison*". Ambrose Pare (1510 – 1590 B.C.) – Against the Unicorn and Bezoar stones. William Piso (1611 – 1678 B.C.) first to recognize the emetic property of Ipecacuanha.[8]

20th century milestones in the development of medical toxicology:

1927 - Federal caustic poison Act.[8]

1938 - Federal food, drug and cosmetic Act.

1949 –First toxicology wards open in Prude pest and Copenhagen.

1949 –First poison information service started in Netherland.

1953 - First U.S. poison center was opened in Chicago.

1957 –17 poison control centers were opened in Chicago.

1957 – National clearing house for poison control center established.

1958 – American association of poison control **(AAPC)** started.

1960 – Federal hazardous substance Libeling Act.

1962 – 462 poison control centers in U.S.

1968 – American academy of clinical toxicology established **(AACT).**

1972 – Introduction of Microfiche Technology for poison information.

1974 – American board of medical toxicology established **(ABMT).**

1978 – 661 poison control center in U.S

1983 – First examination given for specialist in poison information.

1983 – Introduction of CD-ROM data.

1985 – American board of Applied Toxicology **(ABAT)** established.

1989 – 99 poison control centers (including 36 regional centers) in U.S.

1992 – Medical toxicology recognized by American Board of medical Specialty.

1994 – Poison information center, AIIMS – New Delhi – India.

2004 - Indian society of Toxicology. (IST) (November – 2004). [8]

Few of the most consequential & historically important toxins related disasters are:

Gas Disaster

A toxic gas leak at Union Carbide pesticide plant, Bhopal, India in 1984 when 24,000 kg of methyl isocyanide gas was released accidentally lead to the death of 2500 people.[8]

Food Disaster

England Epping chemical food poisoning in 1956, where a source of food got contaminated when the chemical accidentally spilled over while transporting to a bakery. This outbreak of toxic hepatitis was known as **Epping Jaundice**.[8]

In 1981, an oil contamination epidemic due to consumption of highly macerated covering oil in Spain was responsible for a mysterious poisoning epidemic that affected more than 19,000 people, resulting in 340 deaths.[8]

Therapeutic Drug Disaster

In 1982 in Chicago, a different type of drug disaster resulted from the deliberate tampering of Acetaminophen preparations.[8]

Alcohol and illicit Drug Disaster

"Jake" was a popular ethanol substitute in Southern & Midwestern United states in early 20th century which was sold adulterated with castor oil. [8]

Occupational Toxin Epidemics

With the industrial revolution in 19th century, a peculiar disorder attributed to the affects of inhaling mercury vapor was described among the glass manufactures in New Jersey, 5% of hatters died from renal failure.[8]

Exposure to Asbestos in 20th century became the largest and the worst accidental & environmental disaster of all time. Workers sustained asbestos related disease-Lung cancer and Asbestosis.

MOST COMMON POISON ENCOUNTERED IN THIS LOCALITY0

PESTICIDES

Pesticides are the compounds used to kill pests (Insects, rodents, fungi, herbs etc.

Pesticides are classified as:

1. **Insecticides** these are the compounds used to kill or repel insects and related species. Examples are – organophosphorous compounds, Carbamates, Organochlorines, Pyrethroids.
2. **Rodenticides**: these are the compound used to kill rodent like rat, mice, mole etc. Examples are – Zinc phosphide, barium carbonate, strychnine, warfarin etc.
3. **Herbicides**: these are the compounds used to kill weed. Examples are acrolein, glyphosate, paraquat etc.
4. **Fungicides**: these are the compounds used to kill fungi and moulds. Examples are – thiocarbamate, sodium azide.
5. **Miscellaneous compounds** include lead, copper, mercury, nicotine etc. [4]

Category

LETHAL DOSE: The dose of a chemical or biological preparation that is likely to cause death, when followed by a subscript (generally "LD_{50}" or median lethal dose), it denotes the dose likely to cause death in a certain percentage (50%) of the test animals; median lethal dose is LD_{50}, absolute lethal dose is LD_{100}, and minimal lethal dose is LD_{05}

Extremely Toxic or Highly Toxic.

Extremely Toxic : LD50:1-50mg/kg.

Highly Toxic : LD50:51-500mg/kg.[4]

Moderately Toxic or Slightly Toxic.

Moderately Toxic : LD50:501-5000mg/kg.

Slightly Toxic: LD50: > 5000mg/kg.[4]

Colours of Identification based on Labels (insecticide rules 1971)

Extremely Toxic - **Bright Red.**

Highly Toxic- **Bright Yellow.**

Moderately Toxic - **Bright Blue**

Slightly Toxic- **Bright green.**

If it is inflammable it should be mentioned on the label.

Labeling should contain the following: Safe, Nonpoisonous, Non injurious, Harmless. [4]

ORGANOPHOSPHROUS COMPOUNDS

Organophosphorus poisoning is the most common poisoning in India. It is available as dust powder, liquid and they are classified as follows.[4]

Alkyl compounds – such as Tetra Ethyl PyroPhosphate (TEPP), Hexa Ethyl TetraPhosphate (HETP), Octa Methyl PyroPhosphate (OMPA), Malathion etc.

Aryl compounds – such as Parathion, Chlorothion, Diazinon (Tik – 20), Paraoxon etc.

Absorption, Metabolism and Excretion

Organophosphorus compounds are absorbed by any route – skin, conjunctiva, inhalation, oral or by direct injection. Some compounds such as parathion are stored in body fat and are released slowly in the circulation, prolonging the duration of toxic action. Parathion is first metabolized to paraoxon, which is the active toxic agent, then to paranitrophenol, excreted in urine. Malathion is metabolized in the liver by esterase; part of this product is excreted in urine as phosphate.

Mechanism of Action

Organophosphorus compounds are inhibitors of acetyl cholinesterase. Acetyl cholinesterase is required to hydrolyze acetylcholine to choline and acetic acid. There is accumulation of acetylcholine with continued stimulation of local receptors and eventually paralysis of nerve or muscle. Organophosphorous intoxication leads to characteristic end-plate abnormalities that reflect the degree of Acetyl cholinesterase inhibition and increase in Acetyl choline concentration at the neuromuscular junction.

Fatal dose:

- TEPP **100 mg,** Parathion **175 mg,** Malathion about **60 gm,** HETP **350 mg,** Diazinon **1 gm.**

Fatal period: 24 hours.

Clinical Features

Acute poisoning – acute peripheral and central cholinergic block.

An intermediate syndrome with weakness.

A delayed distal polyneuropathy.

Acute Poisoning

Muscarinic effects

Excessive salivation, lacrimation, urination, diarrhea, emesis.

Chromolacryorrhea (shedding of red tears) due to accumulation of porphyrin in the lacrimal glands is seen very rarely.

Eyes – miosis, blurring of vision or dimness of vision. Miosis develops due to the inhibition of cholinesterase and marked parasympathomimetic stimulation of iris.

Heart – slow pulse, hypotension.

Urinary bladder – Frequency of micturition, urinary incontinence.

Nicotinic effects

Muscular weakness, adrenal medulla hyper activity, tachycardia, abdominal cramps, hypertension.

CNS effects

Irritability, Apprehension, Restlessness, fine tremors of hands, eyelids, face or tongue, muscular weakness. Convulsions, confusion progressing to stupor to coma, depression of respiratory and circulatory centers.

Modes of Death: Respiratory failure cerebral hypoxia, Hyperthermia, Hepatic failure, Renal failure.[4]

Difference between intermediate syndrome and delayed polyneuropathy

Variable	**Intermediate syndrome**	**Delayed polyneuropathy**
Time of onset after poisoning	1-4 days	2 to 3 weeks
Site of weakness Limb muscles	Proximal	Distal
Neck muscles	Present	Absent
Cranial nerves	Present	Absent
Respiratory muscles	**Present**	**Absent**
Electromyogram	Tetanic fade	Denervation
Recovery, from time of onset	4 to 18 days	6 to 12 months
Organophosphorous compound commonly involved	Fenthion, dimethoate, monocrotophos	Methamidophos, trichlorphos, leptophos

Diagnosis

Cholinesterase level

Depression of RBC cholinesterase level less than 50 percent of normal indicates organophosphorus poisoning. The decrease is due to binding by phosphate group of pesticide. It is a better parameter than plasma cholinesterase.

Depression of plasma (Serum) cholinesterase activity less than 50% of normal indicates Organophosphorus poisoning. This test is not as specific as RBC cholinesterase activity.

Management

Decontamination

Skin – the affected part should be washed thoroughly with water.

Ocular – copious irrigation with normal saline or tap water.

Ingestion – gastric lavage and administration of activated charcoal.[4]

Antidote Administration

Atropine is a competitive antagonist of acetylcholine and blocks muscarinic manifestations of Organophosphorus compounds. It does not reverse peripheral muscular paralysis, which is a nicotinic action.

Atropine should be given 2 mg intravenous, repeated every 10 minutes till pupil dilates (up to 200 mg can been administered in a day). Some authorities recommend administration of atropine until bronchial and other secretions have dried. According to them pupil size and heart rate cannot be used as end-points. Continued treatment with maintenance doses may be required for 1 to 2 weeks.

Oximes are used as they helps to regenerate acetyl cholinesterase at muscarinic, nicotinic and CNS sites. Pralidoxime (2-PAM) is given intravenously as 500 mg/20 ml infusion upto 1 to 2 gm (children 20 to 40 mg/kg).

Supportive measures

Oxygen administration, ventilator assistance, Maintenance of vital parameters, hydration, and urine output. Convulsions should be controlled with judicious use of diazepam

Avoid Giving

Physostigmine, endorphonium, Succinylcholine for rapid intubation.

ORGANOCHLORINES

Organo chlorine insecticides are chlorinated hydrocarbons and are divided further as:

DichloroDiphenyl-Trichloroethane (DDT) and analogues.

Benzene hexachloride group – e.g. BHC, endrin, aldrin, dieldrin, endosulfan,

Toxaphene and related compounds – e.g. toxaphene.

Availability

Dusting powder, Emulsion, Granules, Solutions

Fatal Dose

DDT - 15 to 30 gm; Lindane - 15 – 30 mg; Aldrin, Endrin, Dieldrin - 2 to 6gm.

Absorption, Metabolism and Excretion

Organochlorine compounds are absorbed through skin, inhalation and through gastrointestinal tract and remains in the tissues like fat, for prolonged duration. These compounds are metabolized in liver excreted in urine, faeces and milk.

Mechanism of Action

Organochlorines affect nerve impulse transmission by altering membrane Na^+ and K^+ flux, resulting in CNS hyper excitability and produce myocardial irritability, predisposing to cardiac arrhythmias.

DDT and related compounds affect the Na+ channel and its conductance across the neuronal membrane, especially of axons.

Cyclodine and Lindane appear to inhibit the GABA - mediated chloride channels in the CNS.

Clinical Features

Acute poisoning

Nausea, vomiting, diarrhoea, hyperaesthesia or paresthesia of mouth and face, headache, vertigo, myoclonus, mydriasis, weakness, agitation, confusion, coma, cough, wheezing, in case of aspiration or inhalation, renal failure, hepatitis, dermatitis.

Chronic poisoning

Exposure of these compounds for prolonged duration may result in cumulative toxicity characterized by anorexia, weight loss, weakness, tremors, ataxia, oligospermia, thrombocytopenic purpura. Lindane and BHC have been linked to aplastic anemia.

Management

Skin – the affected part should be washed thoroughly with water, Ocular – copious irrigation with normal saline or tap water, Ingestion – gastric lavage and administration of activated charcoal, Oxygen administration, ventilator assistance, vital parameters, hydration, and urine output should be maintained, convulsions should be controlled with judicious use of diazepam or lorazepam, arrhythmias can be managed with lidocaine.

Avoid Giving

Epinephrine, Atropine, Oil based fluid/food/cathartics.

Medico legal Importance

Suicidal poisoning is common with this insecticide. Accidental poisoning may occur in farmers while spraying in fields or opening the lid of the containers. Homicidal poisoning is rare as it is difficult to mask the smell of insecticide. [4]

CARBAMATE

Carbamates are popular insecticides and include Aldicrab, Propoxur (Baygon), Carbaryl, Carbofuran, Methomyl, Triallate, Bendiocarb etc.

Mechanism of Action

Carbamate causes reversible inhibition of acetylcholinesterase causing an accumulation of acetylcholine at muscarinic and nicotinic receptors in the CNS.

Fatal Dose

Extremely toxic or highly toxic – Carbaryl, Carbofuran, Methomyl, Propoxur.

Moderately toxic or slightly toxic – Aldicarb, Triallate.

Clinical Features

Clinical features of Carbamate poisoning are similar to Organophosphorous poisoning with following differences. Carbamates causes reversible inhibition of acetyl cholinesterase.. Carbamate toxicity is short lived and it is hydrolyzed spontaneously from the site. It does not penetrate effectively in the CNS so it produces little or no CNS toxicity.

Management

Decontamination : Skin – the affected part should be washed thoroughly with water, Eyes– copious irrigation with normal saline or tap water, Ingestion – gastric lavage and administration of activated charcoal.

Antidote Administration

Atropine is competitive antagonist of acetylcholine and blocks muscarinic manifestations. The atropine should be given 2 mg intravenously with the dose repeated every 10 minutes till signs of atropinization are evident.

Supportive measures

Oxygen administration, ventilator assistance, maintain vital parameters, hydration, urine output

Medico legal Importance

Suicidal poisoning is also common with this insecticide, accidental poisoning may occur, homicidal poisoning is rare as it is difficult to mask the smell of this insecticide. [4]

PYRETHRUM, PYRETHRINS AND PYRETHROIDS

Pyrethrum is extract of the chrysanthemum flower. Pyrethrum contains six active components labeled pyrethrins. Pyrethroids are synthetic derivatives of pyrethrins.

These compounds are commonly used as insect and mosquito repellants.

Mechanism of Action

Pyrethroids prolong the inactivation of the sodium channel by binding in the open state. These compounds quickly inactivate insects but mammals are relatively resistant to them. However, in most of the cases, toxicity occurs because of the allergic reactions to these compounds.

Fatal Dose

Pyrethrum - 1 gm/kg.

Clinical Features

Dermal exposure causes erythema, dermatitis, blister formation, Ocular exposure causes irritation, lacrimation, Inhalation causes rhinorrhoea, sore throat, wheezing, cough, and dyspnoea[9.]

Management

Decontamination: Skin – the affected part should be washed thoroughly with water.

Eyes – copious irrigation with normal saline or tap water.

Systemic Poisoning

Ingestion – gastric lavage and administration of activated charcoal, Fatty substance should be avoided as they prompt the adsorption through GIT, allergic reaction treated with epinephrine and antihistamines. Bronchospasm should be treated with bronchodilators, convulsions should be controlled with judicious use of diazepam. Oxygen administration, ventilator assistance if required. [9]

Medico legal Importance

Suicidal poisoning is rare, Accidental poisoning may occur.[9]

Zinc Phosphide

Physical Appearance

Zinc phosphide is available as dark grey tetragonal crystals or crystalline powder marketed under various trade names (Agrophos, Commando, Sudarshan, Ratoff, Ratol, Robart, etc). It has a repulsive odour of rotten fish.[9]

Uses: Rodenticide.[9]

Fatal Dose: 2-4 grams.

Mechanism of Action:

When exposed to air and moisture, zinc phosphide liberates phosphine which causes multiorgan damage. Phosphine produces widespread organ damage due to cellular hypoxia as a result of binding with cytochrome oxidase, an important respiratory enzyme.

Clinical features

Common presenting symptoms include metallic taste, vomiting, garlicky (fishy) odour of breath, intense thirst, burning epigastric pain and diarrhoea. In severe cases, there is cardiovascular manifestation such as hypotension, tachycardia / bradycardia, massive focal myocardial injury with elevated serum levels of cardiac enzymes occur. Coma supervenes in later stages. Hepatic damage, renal failure and metabolic acidosis are possible. Respiratory distress is invariably present with cyanosis and cold clammy skin.

Medico Legal Importance

Accidental and Suicidal poisonings have been reported involving the consumption of rat pastes containing zinc phosphide. Some of these brands are marketed in tubes that look very similar to toothpaste tubes leading to accidental use.[9]

OLEANDER

Botanical name: Nerium odorum

Common names: Common oleander, white oleander, kaner. [9]

Toxic Principles: Oleandrin (glycoside), Nerin, Folinerin

Mechanism of Action

The glycosides have digoxin like action and inhibit sodium potassium ATPase

Fatal Dose

15 to 20 grams of root

5 to 15 leaves

Fatal period: 20 to 36 hours

Clinical Features

Gastrointestinal, cardiac symptoms accompanied with Delirium and drowsiness

Management

Gastric lavage with activated charcoal. Atropine for AV block and sinus tachycardia and Symptomatic management.

YELLOW OLEANDER

Botanical name: Cerbera thevetia

Common name: Yellow oleander, pila kaner, exile.[9]

Toxic principles: Thevetin, Thevetoxin, Cerberin

Fatal Dose:

- 8 to 10 seeds
- 15 to 20 gm of root
- 5 to 10 leaves

Fatal period: 2 to 3 hours if powdered root taken.

Clinical Features

The milky juice (sap) if applied to skin may cause inflammation in sensitive individuals, Numbness in mouth and tongue, vomiting, diarrhoea, Headache, Giddiness, Cardiogenic shock, Jaundice, Renal failure

Management

Gastric lavage with multiple doses of activated charcoal will promotes its elimination. Bradyarrhythmias are treated with intravenous boluses of atropine and intravenous infusion of isoprenaline.

Medicolegal Importance

Root used for causing abortion, Accidental death occurs due to consumption of folk medicine containing oleander, used as Cattle poison, Poisoning is mostly suicidal, homicide is rare. Common oleander resists decomposition and burning, thus can be detected from decomposed bodies or ash.[9]

DATURA

Common name: Thorn apple, jimson seed

Two varieties of Datura are found in India:

Datura Niger – Deep purple colour flowers.

Datura Alba – White colour flowers.

Toxic part: full plant is toxic and seed is more toxic .

Active Principles:

- Hyoscine (scopolamine), Hyoscyamine, Atropine

Together referred as belladonna alkaloids

Mechanism of Action

The alkaloids competitively inhibit the muscarinic effects of acetylcholine.

Absorption, Metabolism and Excretion

The alkaloids are quickly absorbed from mucous membrane, skin and are excreted by the kidneys.

Clinical Features

The clinical features are best summarized in classical phrase quoted by Morton Still **"Blind as bat, hot as hare, dry as bone, red as beet and mad as hen"**.

Symptoms include, dryness of mouth (dry as bone), bitter taste, dysphagia, dilated pupils, diplopia, difficulty in vision (blurring of vision, blind as bat), dry hot skin with flushing (red as beet), hyperpyrexia (hot as hare), drunken gait (ataxia), convulsions, delirium, agitation, amnesia, incoherence, visual or auditory hallucinations (mad as a hen), Deficit of recent memory. Remote

memory will be undisturbed, dysuria, distention of bladder (retention of urine) and death.

Management

Gastric lavage with activated charcoal, agitation can be controlled with judicious use of diazepam/lorazepam.

Antidote is physostigmine. Intravenous physostigmine is given slowly over 5 – 10 minutes if hyperthermia, delirium, convulsions, hypertension and arrhythmias occurred.[9]

CYANIDE

Cyanide occurs in solid state, liquid state or in gaseous state.

Solid form occurs as salts such as potassium cyanide / sodium cyanide.

Liquid form is Hydrocyanic acid (Prussic acid)

Gaseous form is Hydrogen cyanide (HCN). [9]

Sources

Plants: cyanogenic glycoside

Combustion:

From burnt plastic furniture, Burning of silk or wool, Cigarette smoking.

Mechanism of Action

Cyanide reversibly inhibits ferric iron containing enzymes, causing cytotoxic anoxia

Fatal Dose:

51-100mg - Hydrocyanic acid.

51-80 – of bitter almonds.

200-300mg - Sodium / potassium cyanide.

Inhalation of 1 part in 2000 – Hydrogen Cyanide

Fatal period: is 2-10 minutes.

Clinical features

Inhalation:

Constriction of throat, dizziness, loss of consciousness, coma, and death.

Ingestion:

Nausea, vomiting, abdominal pain, numbness headache, anxiety, agitation, dizziness, confusion, convulsions, coma. Initially bradycardia and hypertension and later tachycardia and hypotension, arrhythmia, tachypnoea followed by bradypnoea.

Skin:

Perspiration, Cherry red colour bullae.

Chronic Poisoning:

Headache, Amblyopia, Optic atrophy, Peripheral neuropathy, Ataxia, Deafness, Glossitis, Stomatitis.

Management

Ingestion

- Gastric lavage is given - 5% sodium thiosulfate solution.
- Sodium nitrate is given as slow i.v.
- Antidote – amyl nitrate – inhalation
- Then Sodium thiosulfate is given 25% solution I.V.

Mechanism of action of nitrites

Nitrites induce methemoglobinemia, Cyanide combines with methemoglobin and form non – toxic cyanmethemoglobin.

Mechanism of action of sodium thiosulfate:

Sodium thiosulfate serves as a substrate for the enzyme rhodanase to catabolise cyanide to non – toxic thiocyanate, which is excreted in the urine.

Medico Legal Importance

Suicide - persons working in chemistry, electroplating, mining and metal heat treatment. Homicide and accidents – rare. **Embalming** interferes with cyanide detection; therefore interpretation in postmortem period becomes difficult.[9]

ALPRAZOLAM

Trade name – Alprax, Trika

It is a very potent benzodiazepine. Orally taken bioavailability is 80-100%.

The Pharmacology of Alprazolam

It has a very potent benzodiazepine. It causes significant suppression of hypothalamic-pituitary-adrenal axis. It has an overall inhibitory effect on nervous activity, by triggering the conductance of chloride ions in the target cell.

Effects of Alprazolam

1. It relieves anxiety & feel 2.for sleep & sedation.

CHLORPHENIRAMINE MALEATE

Trade name – Avil

It belongs to Antihistamines group. (H_1 Receptor Antagonists) [4]

Uses

Treatment of allergic reactions and allergic disorder, Symptomatic relief of common cold, treatment of Vertigo, travel sickness, Anti emetic, Sleeping aid.[4]

Adverse Effects (Therapeutic Doses)

Drowsiness, decreased bowel sounds, tachycardia, urinary retention, Nausea, dystonic reactions, vomiting, hepatotoxicity, chest tightness , Pneumonitis, mydriasis, visual disturbances and wheezing.[4]

Clinical features

Sinus tachycardia with hypo/hypertension, dryness of skin and mucous membranes, cutaneous flushing, anhydrosis, hyperthermia, urinary retention, vomiting, diarrhea, constipation. Rhabdomyolysis can occur.[4]

Management

If less than four times the maximum daily dose has been ingested by an asymptomatic patient, he may be observed at home.

Stomach wash with activated charcoal. Whole bowel irrigation with polyethylene glycol electrolye lavage solution in patients with extremely large ingestions and those involving sustained release preparations..

Physostigmine for anticholinergic effects 2mg for adults, 0.5mg for children by slow i.v. It can be repeated at 5-10 minute intervals if there is no significant improvement. Diazepam i.v for agitation, psychosis, or convulsions. If seizures

persist or recur administer phenobarbitone. Monitor for respiratory depression, hypotension, arrhythmias and need for endotracheal intubation.

For Sinus tachyarrhythmias a short acting cardioselective agent such as esmolol can be used. Cardiotoxicity necessitates careful cardiac monitoring. Dysrhythmias can be corrected with i.v magnesium sulfate (2-6 grams in adults; 25-50 mg/kg in children) or lignocaine.[4]

MEDICO-LEGAL IMPORTANCE

Apart from the fact that these agents are not commonly involved in accident or several investigators have found that these agents decrease skills. They are therefore not recommended for individuals who drive motor vehicles or operate machinery.

Hallucinations, confusion, disorientation, tachycardia and systolic hypertension appeared to be the most commonly occurring effects.[4]

PARACETAMOL

TRADE NAME: ACETOMINOPHEN, WESTERN CALLED AS TYLENOL

Paracetamol is a widely used drug as over the counter antipyretic and analgesic.. Paracetamol induced liver toxicity is more severe in alcoholics.[4]

Organs affected are- Liver, Heart, Kidney, Pancreas, Organs which contain P450 Cytochrome system.

Systemic Effects that are seen due to Overdose are Hepatotoxicity, Metabolic acidosis, Renal toxicity, Cardiac problems.

Fatal Dose: **20-25grams**

Fatal period: **2-4 days**.

Treatment:

Gastric lavage with activated charcoal. **N-acetylcysteine** is a specific antidote and has maximum efficacy if used within 8 hours.

COPPER SULPHATE

Pure metallic copper is not poisonous but "salts of copper" are poisonous.

Uses

Insecticide. Fungicide. Algaecide. Alloy industry [9]

Salts of copper

Chemical name	**Common name**	**Features**
Copper sulphate	Blue vitriol, Nila tutia,	Crystalline blue powder
Copper Subacetate	Verdigiris Zangal	Crystalline green powder
Copper acetoarsenite	Emerald green	Crystalline green powder
Copper arsenite	Scheele's green	Crystalline green powder
Copper Carbonate	Mountain green	Crystalline green powder

Absorption, Metabolism and Excretion

Copper is a normal constituent of body and normal content is 150 mg. It is present in two forms- bound with albumin and other form bound with enzyme ceruloplasmin. Copper is absorbed through bile and traces are found in saliva and milk.

Clinical Features

Acute poisoning

Ingestion

Metallic taste, increased salivation, Colicky abdominal pain, Nausea and vomiting. Vomitus is bluish or greenish in colour. myalgia , Pancreatitis, Methemoglobinemia, haemolysis, Jaundice, oliguria, renal failure, Convulsions, delirium and Coma.

Inhalation of Fumes or Dust Causes

Respiratory tract irritation, Cough, Conjunctivitis, Metal fume fever.

Chronic Poisoning

Abdominal pain, Greenish line on dental margins of gum (**Clapton's line**).

Vineyard Sprayer's lung disease: Copper sulphate is used as an insecticide spray in vineyards. During spraying, chronic inhalation of copper sulphate causes this disease, Greenish hair discolouration, **Wilson's disease.**

Management

Gastric lavage with Potassium Ferro cyanide.

Chelation – initially with Dimercaprol 2.5 mg/kg fourth hourly, Intramuscular followed by oral Penicillamine 2 g/day.

Fatal Dose

Copper sulphate – 30 gm.

Copper subacetate – 15 gm.

Fatal Period: 1 to 3 days.

Medico legal Importance

Suicidal poisoning – common. Accidental poisoning may occur in children. Chronic poisoning – industrial hazard. Criminal abortion and Cattle poison.[9]

KEROSENE POISONING

Kerosene – by the distillation of crude oil , mixture of hydrocarbons is produced [4]

Uses

Solvent, degreaser and domestic fuel.

Adverse Effects

Signs of intoxication, dizziness, nausea, vomiting, headache and chemical pneumonitis.

Dermal contact- dermatitis and chronically can lead to tumor.

Management

Removal of the person from the source, airway should be open, intubate if need then symptomatic treatment .[4]

SULPHURIC ACID

Acids / Alkalies : columnar epithelium of stomach are more sensitive to acids and squamous epithelium of esophagus are more sensitive to alkalies.

Synonyms: Oil of vitriol, Oleum, Battery Acid.[4]

It is an Inorganic Mineral Acid. [4]

Physical Appearance

Sulphuric acid is a heavy, oily, colourless, odourless, non-fuming liquid. It is hygroscopic, it has affinity for water with which it reacts violently, giving off intense heat.

Availability

Sulphuric acid is mainly used in two forms:

1. Commercial concentrated sulphuric acid is usually a 93-98% solution in water.
2. Fuming sulphuric acid is a solution of sulphur trioxide in sulphuric acid.

Fatal Dose

20 – 30 ml of concentrated sulphuric acid.

Deaths have been reported with ingestion of as little as 3.5ml.

TOXICOKINETICS

Systemic absorption of Sulphuric acid is negligible.

Mode of Action

Produces coagulation necrosis of tissues on contact.

Clinical features

Burning pain from the mouth to stomach. Abdominal pain is often severe.

Intense thirst, however attempts at drinking water usually provoke retching. The vomitus is usually brownish or blackish in colour due to altered blood ("Coffee grounds" vomit), and may contain shreds of the charred gastric tissue. If there is coincidental damage to the larynx during swallowing or due to regurgitation, there may be dysphonia, dysphagia and dyspnoea.

Tongue is usually swollen, and blackish or brownish in colour. Teeth become chalky white. There may be constant drooling of saliva, indicative of oesophageal injury. There is often acid spillage while swallowing with consequent corrosion of the skin of the face (especially around the mouth), neck and chest. Burnt skin appears dark brown or black. Features of generalised shock are usually apparent.

Chronic exposure

Occupational exposure to sulphuric acid mist can cause erosion of teeth over a period of time, as also increased incidence of upper respiratory infections.

Occupational exposure may contribute to cases of laryngeal cancer.

Diagnosis

Litmus test: The pH of the saliva can be tested with a litmus paper to determine whether the chemical ingested is an acid or alkali. (Turns red in Acid and blue in Alkaline solution).

Fresh stains in clothing may be tested by adding a few drops of sodium carbonate. Production of effervescence is indicative of an acid stain.

If vomitus or stomach contents are available, add 10% Barium chloride. A heavy white precipitate forms which is insoluble on adding 1ml of nitric acid.

Treatment

Treat symptomatically.

Oral feeds: Depending on degree of damage as assessed by early endoscopy. The following is a rough guide.

Mild (Grade – 1): May have oral feedings on first day.

Moderate (Grade – 2): may have liquids after 48-72 hours.

Severe (Grade – 3): Jejunostomy tube feeding after 48-72hours.

Administration of steroids has been shown to delay stricture formation (in animals) when given within 48 hours of acid ingestion, but is generally not recommended because of increased risk of perforation.

Since there is often severe pain, powerful analgesics such as morphine may be needed.

The use of flexible fibre optic endoscopy is now standard practice in the first 24-48 hours of ingestion to assess the extent of oesophageal and gastric damage. If there are circumferential 2^{nd} or 3^{rd} degree burns, an exploratory laparotomy should be performed. If gastric necrosis is present, an oeosophago-gastrectomy may be considered.

Emergency laparotomy is mandatory if there is perforation or peritonitis. If the patient recovers, there may be long term sequale such as stenosis / stricture formation. Follow-up is therefore essential to look for signs of obstruction, nausea, anorexia, weight loss, surgical procedure such as dilatation, colonic bypass, and oesophago-gastrostomy may be required.

Medico-Legal Importance

Accidental poisoning may arise from mistaken identity since sulphuric acid resembles glycerine and castor oil. It is therefore imperative that it is stored in a distinctive bottle, clearly labelled, and kept in a safe place.

Sulphuric acid is a rare choice for either suicide or homicide. In addition to routine viscera and body fluids, a portion of corroded skin should be cut out,

placed in rectified spirit or absolute alcohol and sent for chemical analysis. Stained clothing must also be sent (preservative not necessary).

Vitriolage

This term refers to the throwing of an acid over face or body of individual in order to disfigure or blind him. The motive is usually revenge or jealousy.

Sulphuric acid is mostly used. In fact, any corrosive which is easy to handle may be used, including organic acids, alkalis and irritant plant juices. [4]

BATHROOM CLEANING ACID

Hydrochloric acid

Synonyms: Muriatic acid; Spirit of salts.[4]

Physical Appearance

Hydrochloric acid is a colourless, fuming liquid which may acquire a yellowish tinge on exposure to air. It is actually hydrogen chloride in water.[4]

Uses

Bleaching agent (less than 10% HCL), dyeing industry, metal refinery, flux for soldering, metal cleaner, drain cleaner, laboratory reagent.

Fatal Dose: About 30 – 40 ml.

Diagnosis

Litmus Test: The pH of the saliva can be tested with a litmus paper to determine whether the chemical ingested is an acid or alkali. (Turns red in Acid and light blue in alkaline solution). If an open bottle of concentrated ammonia solution is placed near the stomach contents or vomitus, copius white fumes of ammonium chloride will emanate. Though normal stomach contents contain

hydrochloric acid, this is usually too dilute (0.2-0.5%) to vitiate the value of this test. Corroded areas are more likely to be greyish, and symptoms are generally less severe when compared to Sulphuric acid.

Treatment

Treat symptomatically. Administration of steroids has been shown to delay stricture formation (in animals) when given within 48 hours of acid ingestion, but the practice is generally not recommended because of increased risk of perforation. Administer antibiotics only if infection occurs. Prophylactic use is not advisable unless corticosteroid therapy is being undertaken. Since there is often severe pain, powerful analgesics such as morphine may have to be given.

Medico-Legal Importance

Accidental poisoning may arise from mistaken identity. It is therefore imperative that it is stored in a distinctive bottle, clearly labelled, and kept in a safe place. Hydrochloric acid is a rare choice for either suicide or homicide. In addition to routine viscera and body fluids, a portion of corroded skin should be cut out, placed in rectified spirit or absolute alcohol and sent for chemical analysis. Stained clothing must also be sent (preservative not necessary).[4]

SUPERVASMOL – 33 HAIR DYE POISONING

Super Vasmol - 33 is hair dye commonly used in India. ParaPhenyleneDiamine (PPD) is toxic compound.[4]

Mechanism of Action

PPD - skin irritant. Renal involvement - transient proteinuria to oliguric acute kidney injury (AKI). Death due to acute kidney failure.

Clinical manifestations

Cervicofacial oedema, cola coloured urine, oliguria, muscular oedema, and shock. Hypocalcaemia may occur in the setting of severe rhabdomyolysis or due to sodium EDTA. Patients can develop seizures, which may be due to toxins in dye or as a result of hypocalcaemia.

Treatment

Supportive therapy with dialysis.[4]

Alcohol :

3 categories-

1. Monohydroxy alcohols- ethanol, methanol
2. Dihydroxy alcohol- propylene glycol, ethelyene glycol
3. Trihydroxy alcohol- glycerol , glycerine

Ethanol:

Synonyms (4)

Ethyl alcohol; Grain alcohol.

Source

Ethanol is produced mostly by synthetic production from ethylene. This is mainly by direct hydration process.

Uses

Beverage, Solvent for after-shaves, colognes, mouthwashes, and perfumes. Several antihistaminic, decongestant, multivitamin, and cough syrups contain varying percentage of alcohol. Rectified spirit (90 to 95 % ethanol) is used as a preservative for viscera, for chemical analysis. Ethanol is used to extract

nucleic acids from whole tissue or tissue culture in virtually all biotechnology processes.

Fatal dose

The usual fatal dose corresponds to approximately 6 grams of ethanol/Kg body weight (adult); 3 gm/Kg (child). In terms of blood alcohol, a level in excess of 400 to 500 mg/100 ml is usually considered to be lethal.

Toxicokinetics

Ethanol is toxic by all routes. After Oral intake Maximum or peak alcohol concentration in blood is reached in 30 to 90 minutes following the last drink. The blood alcohol level generally falls at a rate of 15 to 20 mg/100 ml/hr.This may be higher (upto 30 mg/100 ml/hr) in chronic alcoholics.

Mode of Action

Now there are two theories which are gaining popularity.

1. Ethanol acts by enhancing gamaaminobutyric acid (GABA)-nergic function through interaction with GABA A receptors and associated chloride ion channels. However some investigators are not convinced by this theory.
2. Second theory which appears to be more convincing has to do with N-methyl-d-aspartate (NMDA) ligand gated, glutamate receptors. NMDA receptors mediate neurotoxicity by increasing permeability to calcium and regulate neuronal long-term potentiation. Studies demonstrate that in the acute form of ethanol use, NMDA receptor function is inhibited, while chronic ethanol use results in up-regulation of NMDA receptors.[4]

Clinical Features

1. **Acute Poisoning** (Intoxication, Inebriation): Initially, ethanol produces excitement which progresses to loss of restraint, behavioral changes, garrulousness, slurred speech, ataxia, unsteady gait, drowsiness, stupor, and finally coma.
2. **Chronic Poisoning** (Alcoholism, Ethanolism): Alcoholism is a condition in an individual who consumes large amounts of alcohol over a long period of time. It is characterised by a pathological desire for alcohol intake, black-outs during intoxication, withdrawal symptoms on ceasing alcohol intake.

Treatment

1. **Acute Poisoning** (Intoxication, Inebriation): Airway protection, ventilatory support. Activated charcoal is NOT useful. Stomach wash. Thiamine 100 mg IV. Dextrose– *Indications*—If rapidly determined bedside glucose level is less than 60 mg/100ml, or if rapid determination is not available. *Adult*—25 grams (50 ml of 50% dextrose solution) intravenously; may repeat as needed. *Paediatric*—0.5 to 1 gram dextrose per kg as 25% dextrose solution or 10% dextrose solution (2 to 4ml/kg). *Precautions*—Glucose administration should necessarily be preceded by 100 mg of thiamine IV or IM if chronic alcoholism or malnutrition is suspected, to prevent development of Wernicke's encephalopathy. Intravenous fluid and Hemodialysis.

2. **Chronic poisoning i**nclude treatment of withdrawal symptoms and psychiatric management.

Mediclegal importance

1. Crime and alcoholism
2. Medical etiquette
3. Drunkenness and road traffic accidents.

Others:

Diesel oil

Diesel oil is one of the commonly used fuel in India. Oral intake causes internal burns. Symptoms include Esophageal burns, Mouth burns, Upper respiratory tract burns, Breathing problems, Eye irritation, Skin irritation, Impaired vision, Abdominal pain, Nausea, Vomiting, Blood in vomit, Blood in stool, Reduced blood pressure, Collapse. Treatment is like chemical poisoning that is Decontamination, symptomatic and supportive measures[4.]

After shave lotion

Aftershave is a lotion, gel, or liquid that you can apply to your face after shaving. It's most often used by men. If swallowed, aftershave can produce harmful effects. This is known as aftershave poisoning. Most aftershaves contain isopropyl alcohol (isopropanol) or ethyl alcohol. These ingredients are poisonous when swallowed. Other ingredients vary by brand and product. Aftershave poisoning usually occurs in small children who accidentally drink aftershave. Some people who suffer from alcohol abuse may also drink aftershave when other alcohol is unavailable to become intoxicated[4.]

Sewer gas:

Sewer gas- **(hydrogen sulphide, ammonia, carbonmonoxide,methane)** Decay of organic sulfur-containing products such as fish, manure, sewage, septic tank contents, etc. Poisoning by this gas is almost always accidental, especially in sewer workers. It does not combine with haemoglobin but does so with methhaemoglobin to form sulphmethaemoglobin. Inhalation of this gas causes sudden collapse and respiratory failure[2]

DEMOGRAPHIC PROFILE

15 Suicides is taking place every one hour in India during the year 2014. Suicides in the year 2013 - 1,34,799; in 2014 -1,31,666. Maximum suicides was reported - 16,307 deaths in Maharashtra (12.4%) followed by 16,122 deaths in Tamil nadu state (12.2%) and 14,310 deaths in West Bengal state (10.9%). Among cities, Chennai is leading with 2,214 deaths, then Bengaluru 1906 deaths, followed by Delhi city 1,847 deaths, Mumbai 1,196 deaths, total of the city's contributes 37.0% .suicide rate in the city's is more compared to the all India rate. The prominent means of committing suicides during 2014 - India. Hanging- 41.8%, by consuming poison 26.0%, self-immolation is 6.9%, by drowning 5.6%.Tamil nadu also follows the same order. Total no of deaths due to poisoning in Tamil nadu – in 2014 is 2091[6.]

Death due to Poisoning - a Medico legal Study at Dhaka Medical College, Dhaka.. Out of 300 victims male were 174 (58%) and female were126 (42%). observed in 21-30 years age group 117 (39%) then followed by age group of 31-40 years 84 (28%). OPC was the commonest agent 103 (78.63%) which was followed by alcohol / rectified spirit 12 (9.16%) and then barbiturate 7 (5.34%). According to occupation- agricultural workers or farmers 108 (36%) , housewives 51 (17%). Among the study subjects 178 (59.33%) were illiterate.198 cases (66%) were married. Manner of death 274 victims(91.33%) committed suicide by consuming poisoning and rests 26 (8.67%) were due to accidental poisoning[10.]

The pattern of poisoning in Khammam: Retrospective study- was conducted in Mamata General Hospital Khammam, considering period of 5 years from June –2005 to June –2010 by Bharath K.Guntheti and Uday Pal Singh.[11] 502 cases were studied. It was observed that insecticide poisoning, 390 cases (77.86%) insecticide was the most common poison consumed, followed by zinc phosphide 29 cases (5.778%) and benzodiazepines 16 cases (3.18%), Medicinal drugs 16 cases (3.18), Phenol 9 cases (1.79%), glass pieces 1case (0.19%), Alcohol 1 case (0.19%), kerosene -(0.19%), In 16 cases (3.18%) substance not known. In 23 cases (4.59%) due to snake bite and scorpion sting, 7cases (1.38%). Organophosphorous compounds were the commonest, 372 cases (74.10%), followed by Organochlorine compounds 12 cases (2.39%) and Carbamate 6 cases (1.39%). In 3rd decade of life high incidence were found , that is from 21-30 years, -257 cases (51.19%). Out of 431 males, 341 -males (67.92%) were married and 90 males were unmarried (17.92%). Out of the 71 females, 64 females- (12.74%) were married and 07 females were unmarried (1.39%). Maximum incidence of cases were encountered in summer season 203 cases (40.35%), followed by rainy season 159 cases (31.67%), spring season 82 cases (16.13%) and least during winter 59 cases (11.75). Considering the time of poison intake, 389 Cases -(77.49%) consumed the poison during day time (6A.M to 6P.M) and 113 (22.50%) during night time (6PM to 6 AM). Medium socioeconomic class was the commonest, 398 cases (79.28%) followed by low socio economic class, 96 cases (19.12%) and the least involved was upper class 08 cases (1.59%). With reference to area wise distribution of poisoning, 359

Cases (71.51%) were belonging to rural area and 143 cases (28.48%) belong to urban area. 256 cases were farmers (50.99%) followed by unemployed 112 cases (22.31%), housewives 64 cases (12.74%) and least were students, 26 cases (5.17%). Manner of poisoning- suicidal poisoning 469 cases (93.47%) , then followed by accidental 33 cases (6.57%) and no homicidal poisoning. With reference to mortality, 87cases (17.33%) were due to insecticides, among organophosphorous 72 cases, zinc phosphide 12 cases (13.79%), Organochlorine 6 cases (6.89%) and Carbamate 6 cases (6.89%).

Epidemiological profile of poisoning cases: A hospital based epidemiological study of deaths due to organophosphorus compound poisoning was conducted by S Peranantham1, Kusa Kumar Shaha1, Ajit Sahai2, Siddhartha Das1, G Manigandan1, K Shanmugam, which shows that OPC compound poisoning deaths constitute about 11% of the total number of cases autopsied. The maximum numbers of patients were between 21- 30 years with male predominance, rural background and belonging to lower socioeconomic status. Highest number of poisoning cases are encountered in the daytime (6AM – 6PM).[12]

Sociodemographic profile of poisoning cases: prospective study was conducted in acute poisoning cases admitted in M.K.C.G.- Medical College Hospt., Berhampur, of southern Orissa and fatal cases of poisoning died due to drugs or chemicals during the period from Sep 1999 to Aug 2001 by Shreemanta Kumar Dash, Manoj Kumar Mohanthy, KiranKumar Patnaik, and

Sachidananda Mohanthy. [13] 306 cases were admitted to the Hospital with diagnosis of acute poisoning. Total Males affected were 163 (53.3%) dominating the female. The incidence of poisoning according to age and sex reveals peak incidence in the age group was 21- 30 yrs which represented 124 cases (40.5%). It is evident that 256 cases (83.7%) of victims were of medium socio-economic status. The domicile pattern of the victims shows 178 (58.2%) cases were from rural area, among the 306 cases admitted to the hospital with diagnosis of acute poisoning 156 cases (50.9%) were married. Maximum incidences of cases 97 (31.7%) were recorded in summer season. Considering the time of poisoning, this shows that the majority of cases (55.9%) were observed during the daytime, between 6 AM to 6 PM. organophosphrous is the most commonly abused poison.

Acute poisoning toxiepidemiology: the trend of poisoning cases admitted to the Government District Headquarters Hospital, a secondary care center in Udhagamandalam, Nilgiris District, Tamil Nadu, India, over a 5-year period. 1860 poisoning cases (80 deaths) were reported during the period from Oct 2008 to Sep 2013. The average incidence 1.60 / 1000 population, while the average case fatality rate and mortality rates were 0.07/40.05. The incidence of poisoning increased every year. A total of males 1148. The majority of cases was in 21 -30 age group (41.24%). Suicidal poisoning (*n* = 1,755; 94.35%), next accidental (*n* = 85; 4.57%). Most used was organophosphates**.**[14]

Study the assessment of poisoning cases in rural tertiary Care teaching hospital by a clinical pharmacist: This prospective, study was carried out in

emergency department for a period of 9 months in Adichunchanagiri hospital and research centre by Ramanath.K.V and Naveen kumar HD.[15] A total of 73 cases of Poisoning were admitted during this period. Majority of victims belonged to the age group of 21-40 years (58.9%). Male population was 63.0% and females were 37.0%. The economic status of victims showed low socioeconomic status in 69 cases (94.5%) and remaining 4 cases (5.5%) belongs to medium socio economic status. The marital status of the victims showed married persons were 54.8% followed by single 43.8% and 1.4% widows respectively. The occupation details of victim showed farmers were 38.4%, followed by housewives 21.9%, laborers 17.8%, students 9.6%, businessman 4.1% and others was 8.2%.

Out of 73 cases, 59 cases (80.8%) were intentional, 19.2% cases were observed as accidental poisoning, 10 cases(13.6%) were due to depression, 7 cases(9.6%) were due to financial crisis, 5 cases (6.9%) were due to health problem, 2 cases (2.7%) were due to love issue, 18 cases (24.8) were due to unknown reason and remaining 14(19.0%) cases were due to accidental poisoning. Pesticide exposure was observed in 57.5% cases followed by bites (Snake bite, bee sting) in 13.6%. cases, household products 8.3% cases, medicine 5.6% cases and miscellaneous were 15% cases respectively.

Profile of Poisoning Cases at a North Indian Tertiary Care Hospital: This study was conducted during January-December 2009 from all the poisoning cases admitted during this period to the emergency ward and medical wards in Jawaharlal Nehru Medical College and Hospital, Aligarh, Uttar Pradesh by M.

Shoaib Zaheer, M. Aslam, Vibhanshu Gupta, Vibhor Sharma and Shadab Ahmad khan. [16] Among 104 patients 62 males (59.6%) and 42 females (40.4%) were analyzed in this study. Maximum number of cases (59) were recorded in the age group of 20-29 years. There were 84 cases of suicide (80.8%) that included 52 males and 32 females. There were 16 cases of accidental poisoning (15.4%) which included 9 males and 7 females and 4 cases of homicidal poisoning (3.8%) which included 1 male and 3 female. Aluminium phosphide was the commonest poison consumed (30.8%) followed by zinc phosphide (23.1%) and organophosphorus compounds (15.4%). Other common poisons were sedatives (11.5%) and unknown poisons (11.8%). Overall mortality was 14.4% (15 Cases) while 89 patients had a favorable outcome. Of those expired, maximum mortality was recorded with aluminium phosphide group (37.5%).

Current trends of poisoning - a hospital profile: This statistical study was conducted by Department of Forensic Medicine and Toxicology, Govt. Medical College, Amritsar in the Emergency Wing of Guru Nanak Dev Hospital, from 10-5-1997 to 9-5-1998 by Dr.J.Gargi, Dr. Hakumat Rai, Dr. Ashok Chanana, Dr.Gurmanjit Rai, Dr.Gaurav Sharma, Dr.IJS Bagga.To know the current trend of poisoning in that region.[17] From this study it was observed that aluminium phosphide, 130 cases (38.23%), was very frequently consumed poison followed by organophosphorus compounds 60 cases (17.64%), alcohol 28 cases (8.23%), zinc phosphide 16 cases (4.23%) and 12 cases of food poisoning (3.52%). Rest of the poisons was consumed quite less frequently. Total numbers of cases were 340, among which 248 cases (72.94%) were

males and 92(27.6%) were females. Maximum no. of cases was between 21-30 years. Out of the poisoning cases, 235 cases (69.12%) were married and 105 cases (30.88%) were unmarried. Urban area reported highest incidence of poisoning 175 cases (51.47%) and rural area reported 155 cases (45.59%), whereas 10 cases (2.94%) were of undetermined origin. Suicidal poisoning was highest, 260 cases (76.46%) when compared to accidental 71 cases (20.91%), and homicidal 6 cases (1.76%), 3 cases (0.88%) were undetermined.

Trends of acute poisoning in South Karnataka: This was a Retrospective study of acute poisoning cases admitted to Government Wedlock hospital, Mangalore during the period from 1st January 1999 to 31st December 2003 by Unnikrishnan B, Singh B and Rajeev A.[18] 546 cases brought to Govt Wenlock Hospital in 5 years due to suspected poisoning were studied. In 1999 there were 82 cases of poisoning, which then increased to 141 cases in the year 2002 and then decreased to 118 cases in the year 2003. Males were 380 (69.6%) and females were 166 (30.4%) with the male: female ratio being 2.29:1. Majority (53%) of victims were between 21-40 years followed by the age group between 1-20 years (25%). Most common poison used for suicide purpose was pesticides (Organophosphorous compounds and Organochlorines). 13.3% of the cases were due to snake bite. Among 235 male cases of suicidal poisoning 74% were married and 26% were unmarried. Out of the 126 females 61% were married 39% were unmarried. Unmarried females were more prone to suicide than married females. Suicide by poisoning was most common (68%), while

homicide by poisoning was negligible (0.2%). (31%) suffered due to accidental poisoning.

Trends in Poisoning Deaths : A retrospective study of poisoning cases in Melmaruvathur region of Tamil Nadu for period of 3 years, by O. Gambhir Singh, A. M. Singh. The incidence was highest in the age range from 20-50 years, with mean value of 35 years. Females outnumbered males with a sex ratio of 1: 1.3 (male: female). The incidence was high in married persons. There was not a single case of homicidal poisoning in adults. Most of the patients died within 24hrs of admission[19.]

Acute Chemical and Pharmaceutical Poisoning Cases treated in Civil Hospital, Ahmedabad - One year study: This was a prospective study of poisoned patients admitted to the Emergency Department of Civil Hospital Ahmedabad, from 1st October 2006 to 30th September 2007 by Tejas Prajapati, Kartik Prajapati, Rakesh Tandon, Saumil Merchant.[20] The most common type of poison abused was pesticide in 33.9% of cases followed by household chemicals in 26.8% of cases and pharmaceutical agents in 15.3% of cases. In total 366 cases 70.8% were male. The majority (45.08) of cases had 21-30 years of age. 71.6% of cases lived in rural area and 28.4% of cases were from urban area. Regarding patient occupation, private service workers, housewives, and unemployed persons were the most common. Most patients recovered (81.4%) and the majority of them were discharged during first 4 days of post-admission. 68 Patients died during admission which means case fatality rate was 18.6%.

A study on poisoning cases in a tertiary care hospital: This present study was conducted at the emergency departments and intensive care units (ICUs) of Mahatma Gandhi Memorial Hospital, Warangal, Andhra Pradesh, by Subash Vijaya Kumar, B. Venkateswarlu, M. Sasikala, and G. Vijay Kumar.[21] The study was carried out for the period of 1 year. The patients included in the study were those who had undergone exposure to poison either by household or agricultural pesticides, stings bite, snake bite, industrial toxins, toxic plants, drug or miscellaneous products. The majority of poison cases were between 21 - 30 years of age. There were more male patients than females, with 52.15% males and 47.84% of females respectively. Male poisoning cases were predominantly from rural areas (65%). The overall case fatality rate was 8.3%. The exposure substances identified as most commonly encountered in the emergency department included snakebite 9.3%, Organo phosphorous 17.2%, overdose of drugs 8.4%, scorpion stings 12.5%, unknown pill 7.7%, hair dye 2.6%, corrosive 3%, endosulphan 6.42%, rodenticide 3.1%, kerosene ingestion 2.15%, unknown bite 5.03%, alcohol intoxication 6% house hold item, i.e. All Out, Baygon Spray 2.83%, nail polishes 1.33% and multitablet 12.1%.

The total mortality rate was 8.3%, with males 5.1% and females 3.2% exposure. The findings of the present study revealed a higher incidence of poisoning in males than in females in all age groups. Self-poisoning (suicidal 52%) is the most common manner of acute poisoning, followed by abuse 32%, ADR (Adverse Drug Reaction) 9% and others 6% cases. The results of this study illustrate that a total of 2,226 patients were hospitalized due to acute

poisoning in the hospital. Of these, 186 case (8.3%) patients died due to poisoning.

Profile of poisoning cases in district and Medical College hospitals of north Karnataka: This was a descriptive study of poisoning cases admitted and autopsied at District hospital and Al- Ameen Medical College Hospital, Bijapur during 2004-2005 by Vinay B.Shetty, Gurudatta S.Pawar and P.I.Inamadar.[22] Among 229 cases of poisoning studied majority of the victims were males (57.15%). Male and female ratio was 1.3:1. The commonest age group involved was 21-30 years (42.29%). The commonest type of poison encountered was Organophosphorous compounds (73.14%) and the least was with phenol 1 case (0.57%). The commonest manner of poisoning was suicide both in males (78%) and females (77.33%) followed by accidental poisoning accounting for 21% and 22.67% in males and females respectively. Not a single case of homicidal poisoning was observed in this study.

Profile of suicidal poisoning in Puducherry area conducted by Naveen N, Madhuvardhana T, Arun M, Balakrishna Rao A J, kagne R N, A total of 322 cases reported to during the study period were recorded. Out of this the majority of the victims were female (55.60%), married (53.41%) and the most commonly affected age group was between 21-30years. Most of the victims were unemployed (77.63%), illiterate (19.25%).Nuclear family (86%) and from low economic class (70.6%). Insecticides and Pesticides (41. 92%) were the most common type of poison.[23]

A comparative retrospective study of poisoning cases in Central, Zonal and District Hospitals: This was retrospective study of poisoning conducted at different level of hospitals in Nepal. By Deepak Pokhrel, Sirjana Pant, Anupama Pradhan and Saffar Mansoor. [26] Females were more common to the incidence (51.4%). Subjects between the ages of 14-24 years were mostly prone to the incidence (49%). Rainy season was the season when most of the incidence occurred (31.1%). Married subjects are more prone to the incidence (37.1%) than unmarried (30.6%). Night time was the time when most of the incidence occurred (15.6%). Intentional poisoning (56.4%) was the most common type. Among 229 cases of poisoning studied majority of the victims were males (57.15%). Male and female ratio was 1.3:1. The commonest age group involved was 21-30 years (42.29%). The commonest type of poison encountered was Organophosphorous compounds (73.14%) and the least was with phenol 1 case (0.57%). The commonest manner of poisoning was suicide both in males (78%) and females (77.33%) followed by accidental poisoning accounting for 21% and 22.67% in males and females respectively. Not a single case of homicidal poisoning was observed in this study. Occupation wise agricultural farmers (63cases, 36%) topped the list followed by laborers (23.43%). Most of the victims belonged to rural area 133 (76%). Persons of low socio economic strata are the commonest victims (72.57%) followed by middle class (25.71%) and least involved were the upper class (1.14%).

Epidemiology of poisoning in Fatal cases (Autopsy studies – Sanjay Gupta, S.Kumar & M.I.Sheikh, (Comparative study and changing trends of

poisoning year 2004-2005 at Surat, India). [27] Dr. Karthik Prjapati, Dr. Saumil P.Merchant & Dr. Pratik R Patel. (Trends of Suicidal Poisoning in Ahmedabad (Retrospectivestudy) AMC MET, Medical College, Ahmedabad.) [28.] Naveen Kumar, N.Varma, S.D.Kalele, (Study of Profile of Deaths due to poisoning in Bhavnagar region). [29], Manoj Kumar Mohanthy, Arun.M, etal. (Correlation between postmortem diagnosis and survival time in poisoning Deaths) [30]. Karthik Prajapti, Sanmil P Merchant & Pratik K Patel (Trends of suicidal poisoning in Ahmedabad – A Retrospective study at AMC MET Medical College, Ahmedabad) [31.] B.D.Gupta & P.C.Vazhelp (Profile of fatal poisoning in and around Jamnagar at MP Shah Medical College, Jamnagar) [32]. Krishnan R Siddapur, Gurudatta S Pawar & Shashidahar C Mestri, (Trends of Poisoning and groups stomach mucosal Appearance in Fatal Poisoning stages An autopsy study)[33]. Cyrial Job (A Retrospective study of poisoning cases in Thrissur district of Kerala for the year 1995 at Thrissure Government Medical College – Thrissure) [34]. Dr. Karmjit Singh, Dr.S.S.Oberin, Dr.D.S.Bhullar (Poisoning trends in the molwa region of Punjab at Govt Medical College & Rajindra Hospital), [35].

Epidemiology of Specific Poisoning were also studied like Organophosphorous compounds Alumiumum phosphide, Hair Dye, Benzodiazepines, Alcohol, snake bite – Scorpion, Stings etc – Somasundaram K.V., Ashok Patil, & Shukla S.K. (Epidemiological Profile of OP Poisoning cases treatment at Prawara Hospital, LONI, India),[36]. Amit M Patil, Satin K MESH RAM, Rajesh D kharat, etal., (Profile of Fatal Methyl Alcohol

poisoning outbreak – A Medico legal Autopsy case study [BYL Nair Hospital, Mumbai],[37] Kora S.A, Doddamahi GB, Hal agali G.R.etal., (Sociodemographic Profile of the Organophosphorous poisoning cases in Southern India (S.N.Medical College & Hongal shree kumareshwar Hospital – Bogal kot)[38], Selvam .V, Panneer Selvam.G & Vijayanath.V (Study of Death Incidence by Insecticide poisioning in salem (Government Hospital - Salem).[39] D.G.Gannur, Prakash Maka, K.S.Narana Reddy (Organophosphorous Compound Poisoning in Gulbarga Region – A five years study (Govt Hospital – Gulbarga), Omid Mehrpour, Mandana Polati, Kambiz Soltaninejod, etal., (Evaluation of histopathological changes in Fatal Aluminium Phosphide Poisoning), [TLMC – Tehrah Legal medical Centre –IRAN) [40]. [41] Dr.R.C.Zariwala, Dr.R.S.Bhise, Dr.D.S.Patel, etal., (A retospective study of 5 years: Organophosphourus poisoning in Ahmedabad[Smt. NHLMMC Ahmedaba],[42] Dhaval J.Patel & Pawan R. Tekade (Profile of organophoshphorous poisoning at Mharahi Hospital, J agdalpur, Chathisgarh AThree years study),[43] Hareesh RS Kumar, Raju K, Candran V, etal., (Epidemiological and clinical study of snakebite cases admitted in Basaveshwara Hospital, Chitradurga Karnataka)[44], Dr. S.M.Kar, Dr. Siddhartha Timsinha & Dr. Prasanth Agarwal (An Epidemiology study of Organophosphorous poisoning at Manipal Teaching Hospital, Pokhara, Nepal [Manipal Teaching Hospital, Pokhera – Nepal] [45] Thakur MS, Naik JD Sheetu, MK Jailkhani & Langare SD, (A one year retrospective study of snake bite cases admitted in a tertiary care hospital of western Maharashtra),[46.]Aluminium Phosphide – Ashok Kumar jain, B.P.Dweey,

S.P.Garg, etal.,(Trends of Aluminium Phosphide Poisoning in Bhopal region – A Retrospective study 10 years), [47] Dr. Sarjeev Kumar Chowdary, S.G.Momin, Dipak H.Vara etal., (An Epidemiological study of Fatal Aluminium phosphide poisoning at Rajkot [PDH Medical College & Hospital-Rajkot].[48] Anandakumar l, Sai Pradeep B.V., Sobha Rani.G, etal., (Hair Dye – An Emerging Icon – as a suicidal agent – A short report)[49], P.Bhargava, P.Mathew (Hair Dye Poisoning),[50] Uzma Saleem, Saeed Mahmood, Bilal Ahmad, etal.(Benzodiozepine poisoning cases – A Retrospective study from Faisalabad, Pakistan.) [College of Pharmacy, Government College University Faislabad, Pakistan) – Lahore General Hospital, Pakistan),[51]

Epidemiology of poisoning in Pediatric group – Pratik Vijay Tarvadi, Shakar.M.Bakkannavar, Manjunaths Hb.etal., (Trends of poisoning among children at Kasturba Hospital, Manipal)[52.] Surjit Singh, Sunil Singh, N.Ksood.etal., (Charging pattern of child hood poisoning (1970-1989): Experience of a large North Indian Hospital), [53] Vikrim Palima & G.Pradeep Kumar, (Poisoning deaths in Children).[54] S.Gupta, Y.C.Govil, P.K.Mishra, etal., (Trends in Poisoning in children: Experience at a large referral teaching hospital.),[55] T.Brojen Singh, K.Romeo Singh, Lallan prosod.etal., (Childhood poisoning – A three years experience in RIMS, IMPHAL.). [56] Nowneet Kumar Bhat, Minakshi Dar, Sohad Ahmad, etal., (Profile of poisoning in children and adolescents at a North Indian Tertiary Care Centre). [57]

MATERIALS AND METHODS

Material for my study comprises of 228 cases of death due to poisoning autopsied in Govt Kilpauk Medical College Hospital Chennai was studied. All case records department MRD were studied, over a period from January 2013 to December 2015.

The data consists of:

1. History of the cases, form 86 and other relevant police documents.
2. Case history papers and other relevant hospital documents of the victims.
3. Postmortem reports of the above said cases.
4. Toxicology report from state Forensic science laboratory Chennai.
5. The statistical analysis of the data was done and presented as results and observations in tabular form, graphs and charts.

INCLUSION CRITERIA:

1. All cases of Deaths Due to Poisoning autopsied in Govt. Kilpauk Medical College Chennai- 10

EXCLUSION CRITERIA:

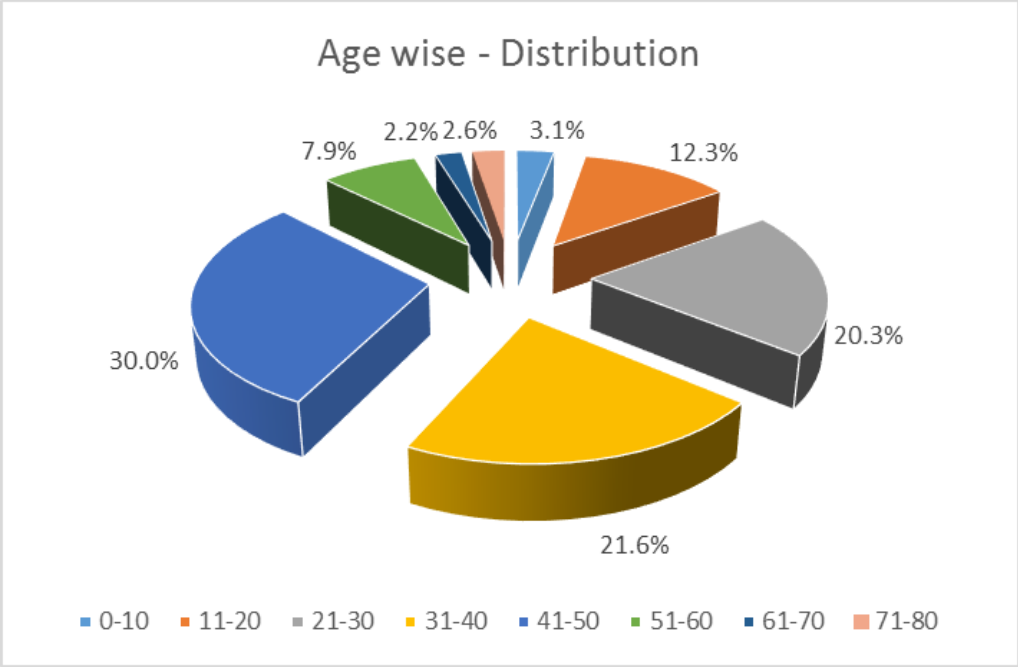
1. Unidentified bodies with no police history of poisoning.
2. Decomposed bodies with no police history of poisoning.

RESULTS AND OBSERVATIONS :

TABLE 1 -AGE WISE DISTRIBUTION

Age	No. of cases	%
0-10	7	3.1%
11-20	28	12.3%
21-30	46	20.3%
31-40	49	21.6%
41-50	**68**	**30.0%**
51-60	18	7.9%
61-70	5	2.2%
71-80	6	2.6%

FIGURE 1 – AGE WISE DISTRIBUTION

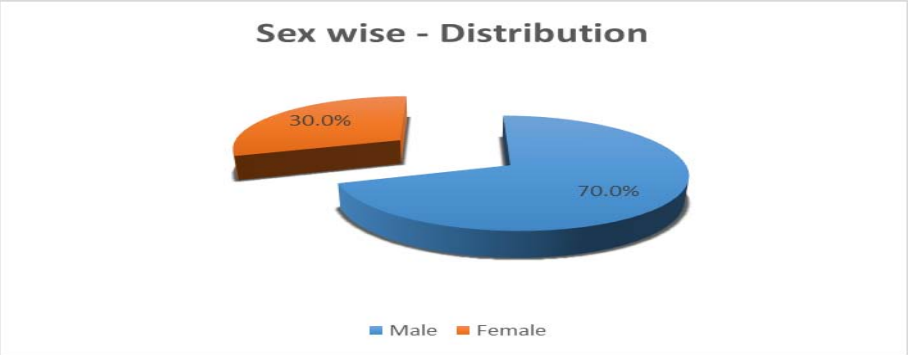


In this study more number of cases were in 41-50 years (68 cases) which is 30.0%

TABLE 2 – SEX WISE DISTRIBUTION

Sex	No. of cases	%
Male	**159**	**70.0%**
Female	68	30.0%

FIGURE 2 – SEX WISE DISTRIBUTION

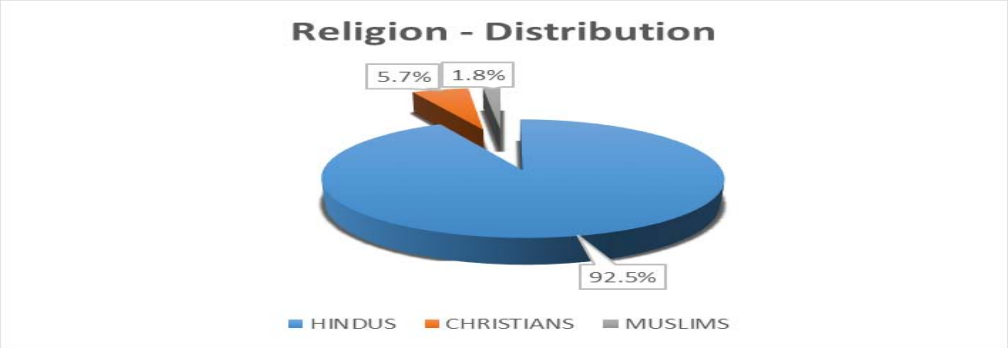


In my study more number of cases (159) was male 70.0%

TABLE 3 – DISTRIBUTION ACCORDING RELIGION

Religion	No. of cases	%
HINDUS	**210**	**92.5%**
CHRISTIANS	13	5.7%
MUSLIMS	4	1.8%

FIGURE 3 – DISTRIBUTION ACCORDING RELIGION

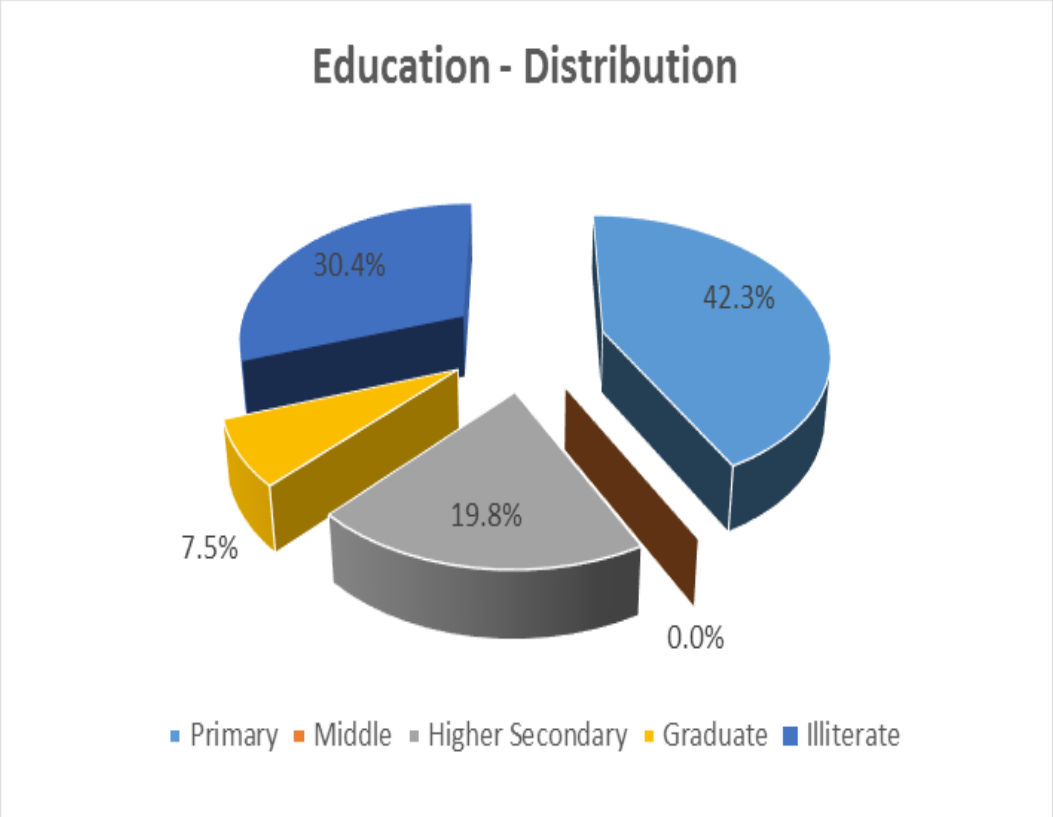


In this study more number of cases belong of hindu religion (210 cases) 92.5%

TABLE 3: EDUCATIONAL STATUS

Education	No. of cases	%
Primary	**96**	**42.3%**
Middle	0	0.0%
Higher Secondary	45	19.8%
Graduate	17	7.5%
Illiterate	69	30.4%

FIGURE 3 : EDUCATIONAL STATUS

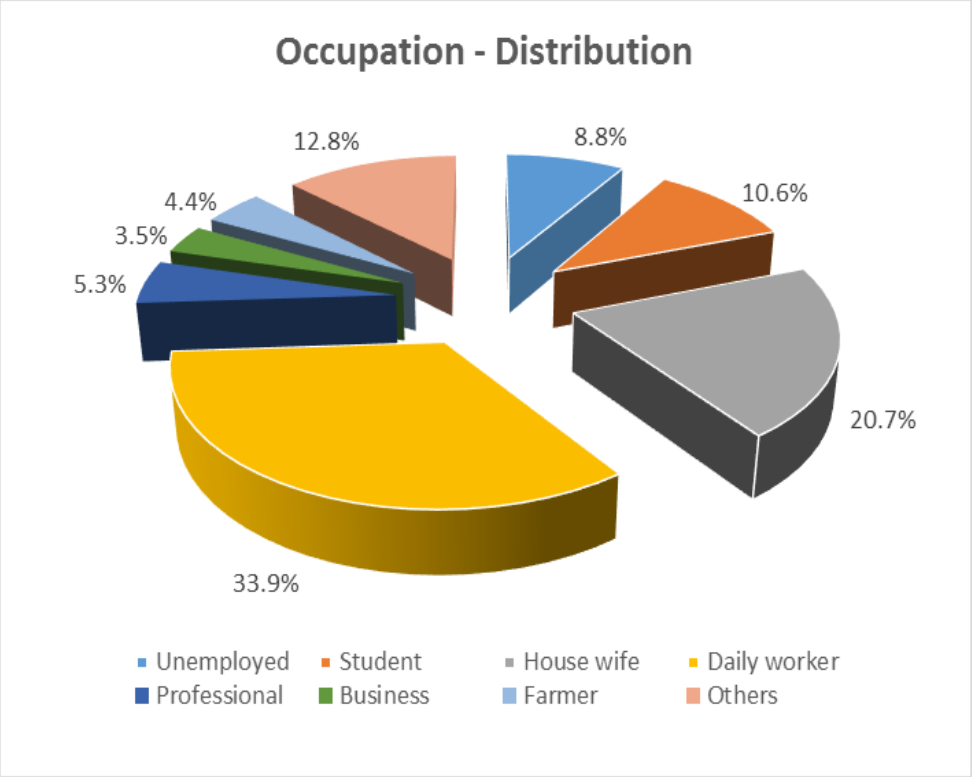


In my study more number of cases (96) studied only till primary education 42.3%

TABLE 5: OCCUPATIONAL WISE DISTRIBUTION

Occupation	No. of cases	%
Unemployed	20	8.8%
Student	24	10.6%
House wife	47	20.7%
Daily wage worker	**77**	**33.9%**
Professional	12	5.3%
Business	8	3.5%
Farmer	**10**	**4.4%**
Others	29	12.8%

FIGURE 5: OCCUPATIONAL WISE DISTRIBUTION

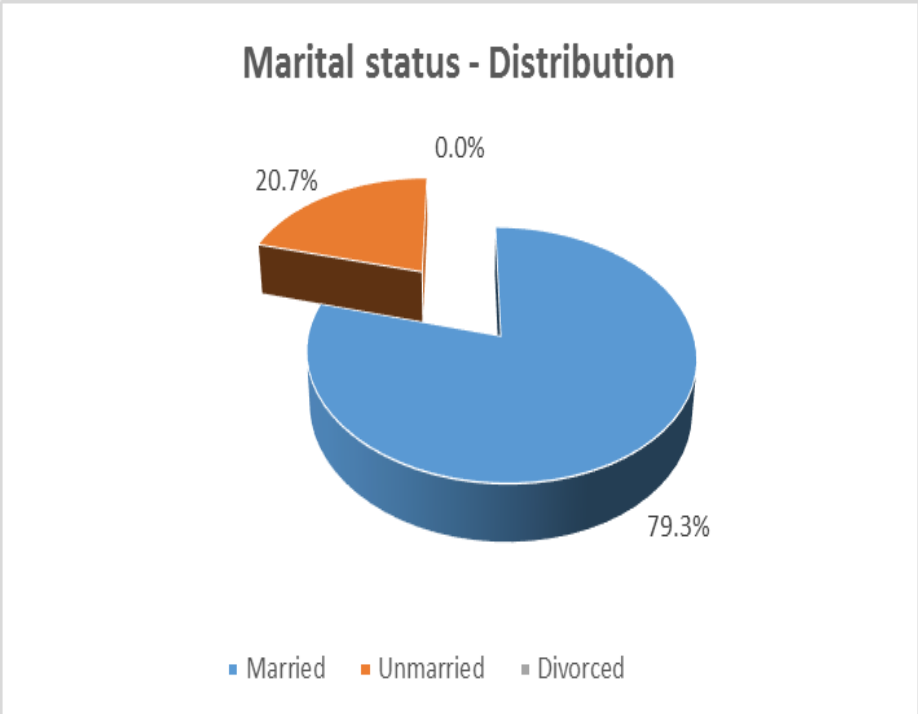


In this study more number of cases (77) were daily wage workers 33.9%

TABLE 6: MARITAL STATUS

MARITAL STATUS	No. of cases	%
Married	**180**	**79.3%**
Unmarried	47	20.7%
Divorced	0	0.0%

FIGURE 6 : MARITAL STATUS

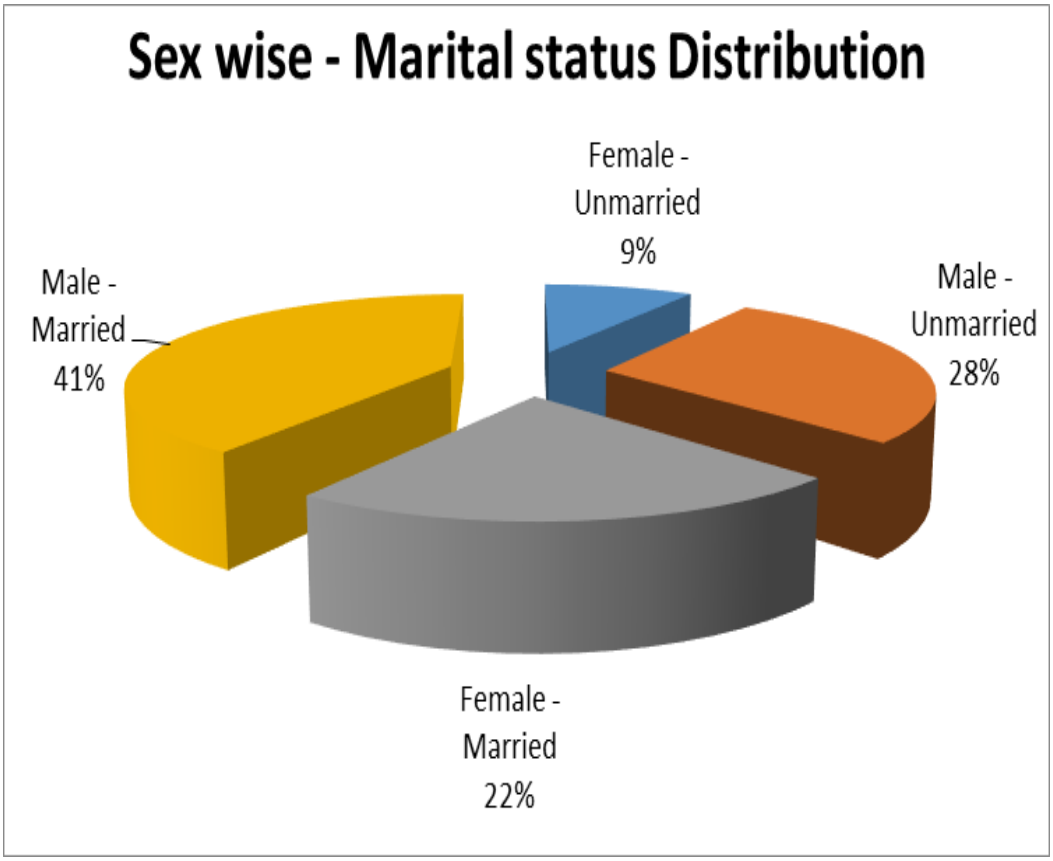


In this study more number of cases were married (180) 79.3%

TABLE 6A: SEX – MARITAL STATUS DISTRIBUTION:

Type	No. of cases	%
Female – Unmarried	21	9.25%
Male – Unmarried	64	28.2%
Female – married	50	22.0%
Male – married	**92**	**40.5%**

FIGURE 6A: SEX – MARITAL STATUS DISTRIBUTION:

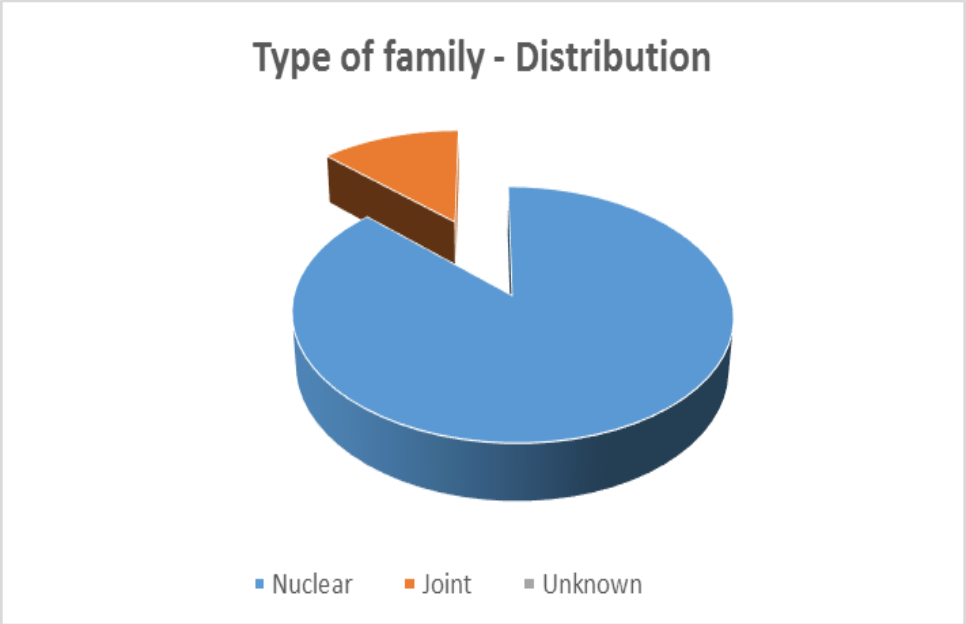


In this study most of the case are Married male (92) 40.5%

TABLE 7: TYPE OF FAMILY

Type	No. of cases	%
Nuclear	**198**	**87.2%**
Joint	29	12.8%
Unknown	0	0.0%

FIGURE 7: TYPE OF FAMILY

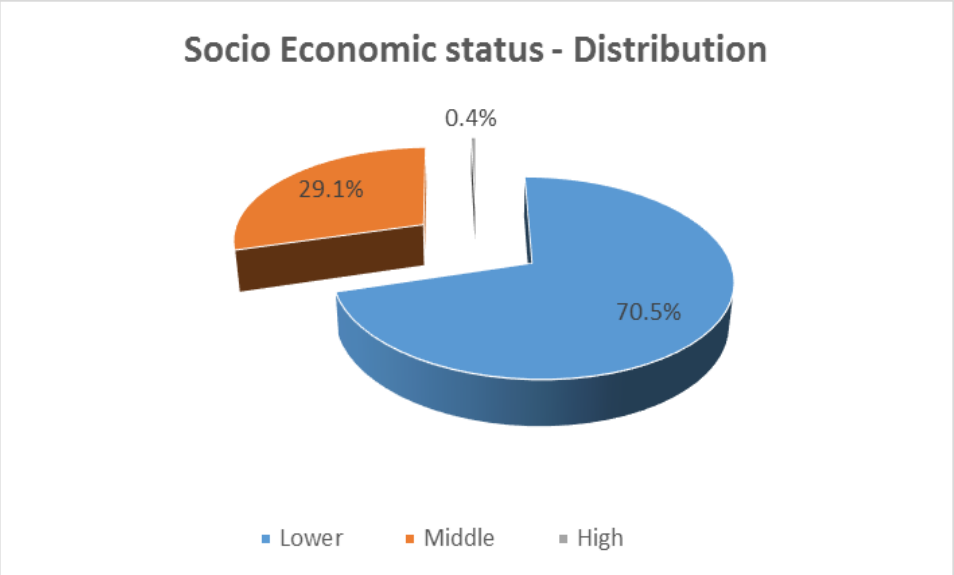


In my study more number of cases belong to nuclear family (198) 87.2%

TABLE 8: SOCIO-ECONOMIC STATUS

Socio-economic status	No. of cases	%
Lower	**160**	**70.5%**
Middle	66	29.1%
High	1	0.4%

FIGURE 8: SCOCIO-ECONOMIC STATUS

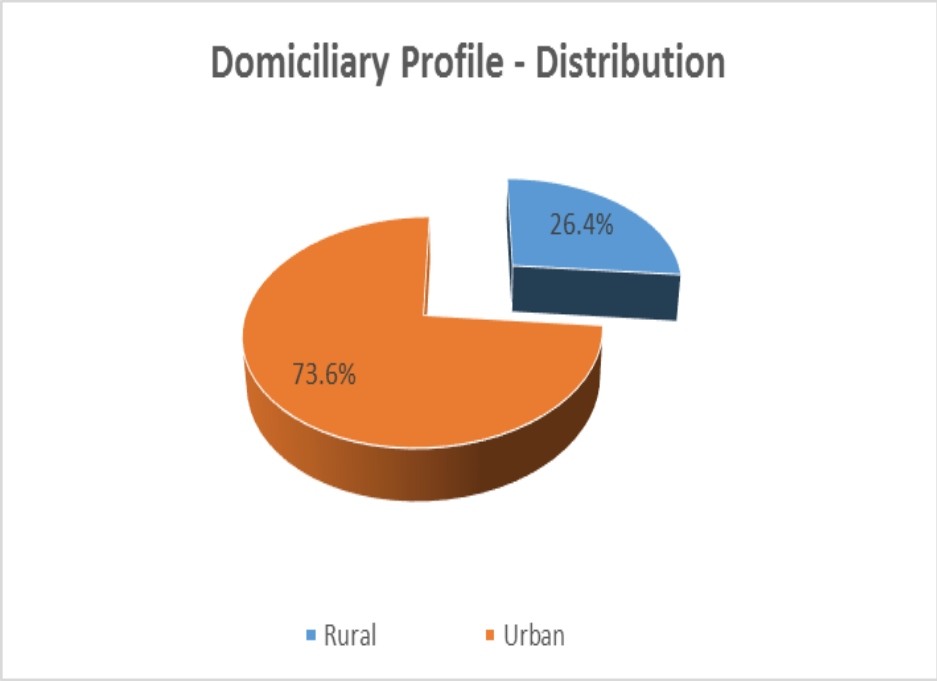


In my study most of the cases belongs to lower socio economic status (160) 70.5%

TABLE 9: DOMICILIARY PROFILE

DOMICILIARY PROFILE	No. of cases	%
Rural	60	26.4%
Urban	**167**	**73.6%**

FIGURE 9: DOMICILIARY PROFILE

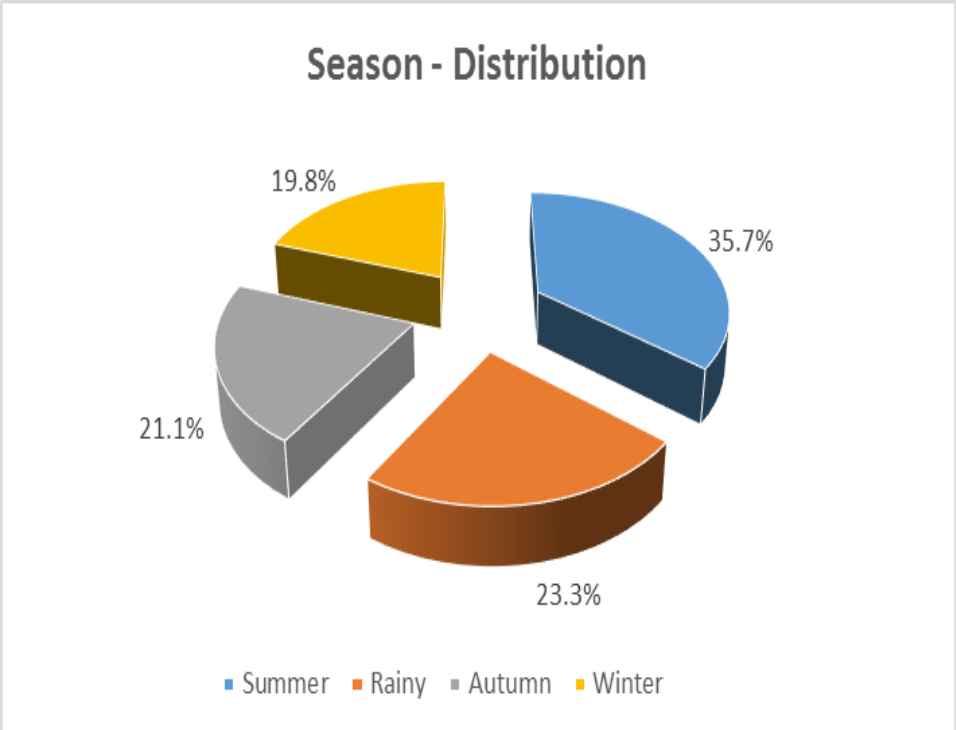


In my study more number of cases are urban dwellers (167) 73.6%

TABLE 10: SESONAL DISTRIBUTION

Season	No. of cases	%
Summer	**81**	**35.7%**
Rainy	53	23.3%
Autumn	48	21.1%
Winter	45	19.8%

FIGURE 10: SESONAL DISTRIBUTION

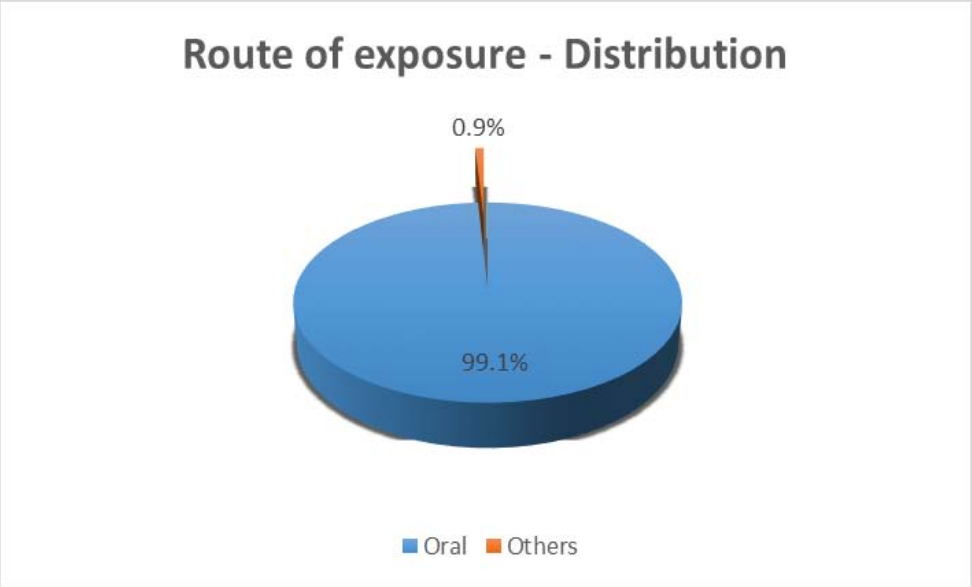


In my study most of the cases consumed poison during summer season (81) 35.7%

TABLE 11: ROUTE OF EXPOSURE

Route of exposure	**No. of cases**	**%**
Oral	**225**	**99.1%**
Others (inhalation)	2	0.9%

FIGURE 11: ROUTE OF EXPOSURE

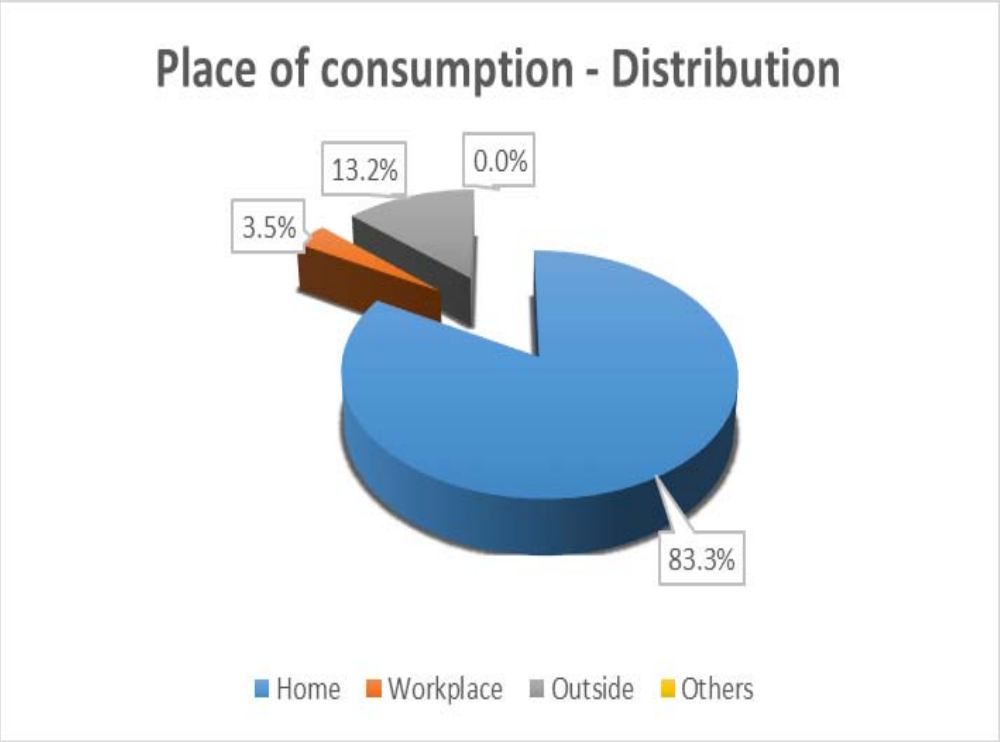


In this study almost all cases consumed poison orally (225) 99.1%. 2 cases died due to inhalation of poisonous sewer gas.

TABLE 12: PLACE OF CONSUMPTION

Place of consumption	No of cases	%
Home	**189**	**83.3%**
Workplace	8	3.5%
Outside	30	13.2%
Others	0	0.0%

FIGURE 12 : PLACE OF CONSUMPTION

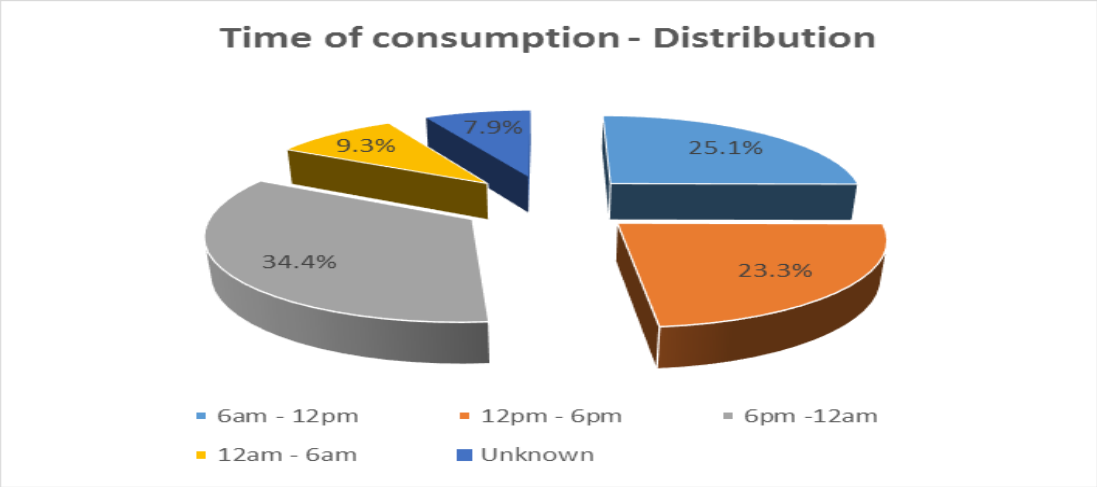


In this study most of them consumed poison at home (189) 83.3%

TABLE 13 : TIME OF CONSUMPTION

Time of consumption	No. of cases	%
6am - 12pm	57	25.1%
12pm - 6pm	53	23.3%
6pm -12am	**78**	**34.4%**
12am - 6am	21	9.3%
Unknown	18	7.9%

FIGURE 13: TIME OF CONSUMPTION

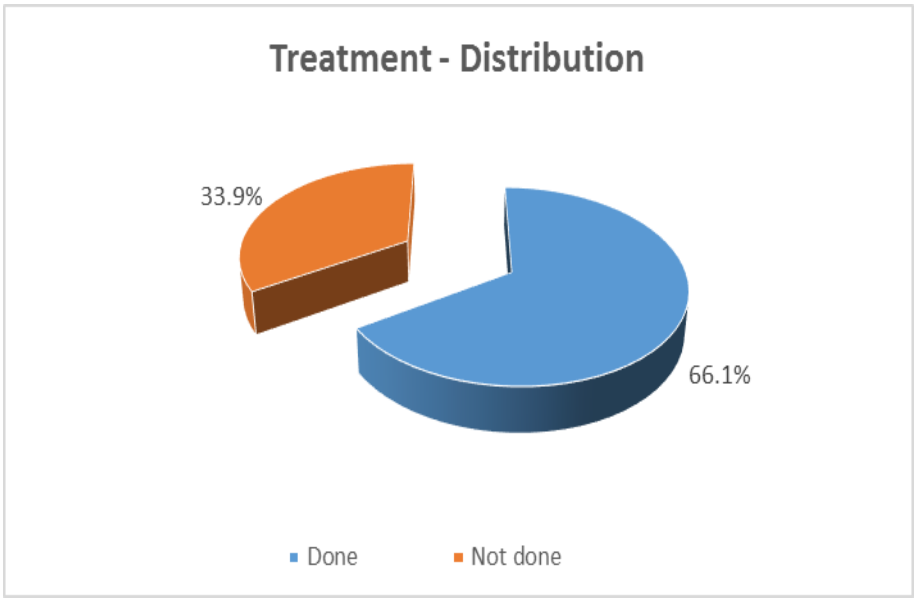


In this study most of the cases consumed poison between 6pm-12am (78) 34.4%

TABLE 14 : TREATMENT

Treatment	No. of cases	%
Done	**150**	**66.1%**
Not done	77	33.9%

FIGURE 14: TREATMENT

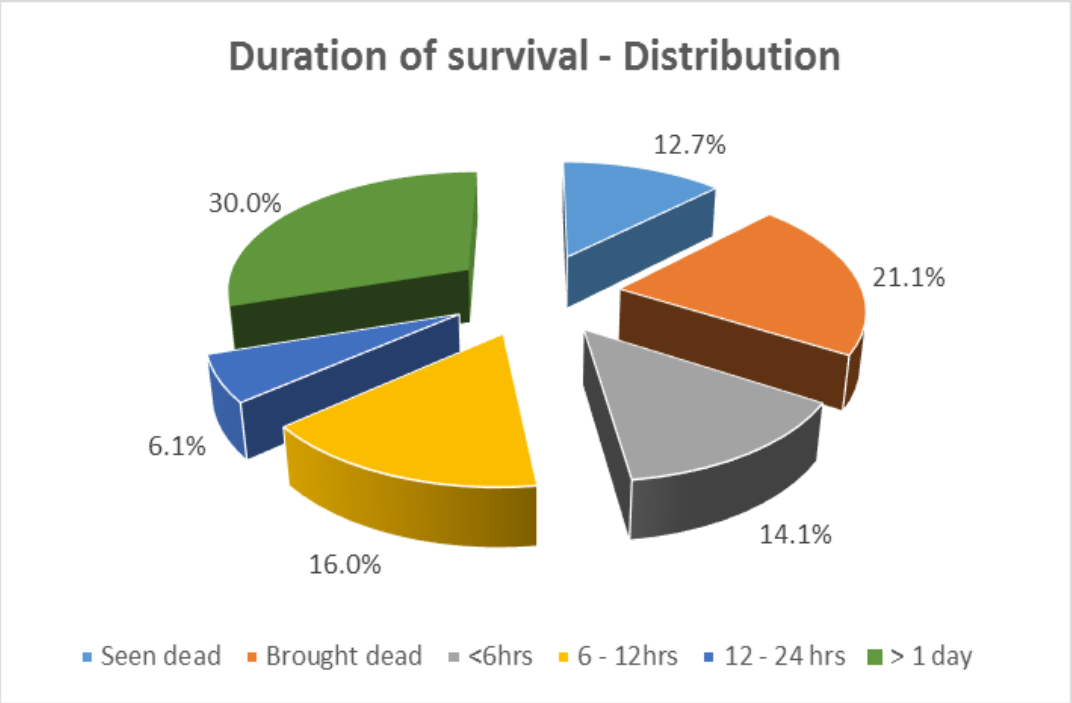


In this study treatment was done in 150 cases 66.1%

TABLE 15: DURATION OF SURVIVAL

Duration of survival	No. of cases	%
Seen dead	27	11.9%
Brought dead	45	19.8%
<6hrs	30	13.2%
6 - 12hrs	34	15.0%
12 - 24 hrs	13	5.7%
> 1 day	**64**	**28.2%**

FIGURE 15: DURATION OF SURVIVAL

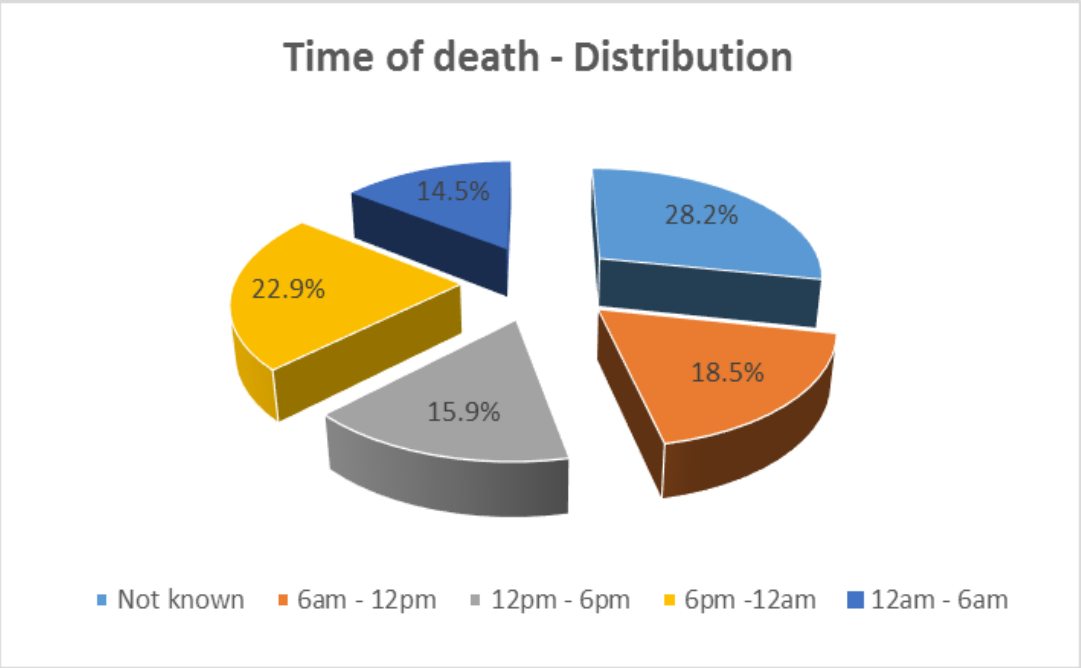


In this study more number of cases survived more than one day (64) 28.2%

TABLE 16 : TIME OF DEATH

Time of death	No. of cases	%
Not known	**64**	**28.2%**
6am - 12pm	42	18.5%
12pm - 6pm	36	15.9%
6pm -12am	52	22.9%
12am - 6am	33	14.5%

FIGURE 16: TIME OF DEATH

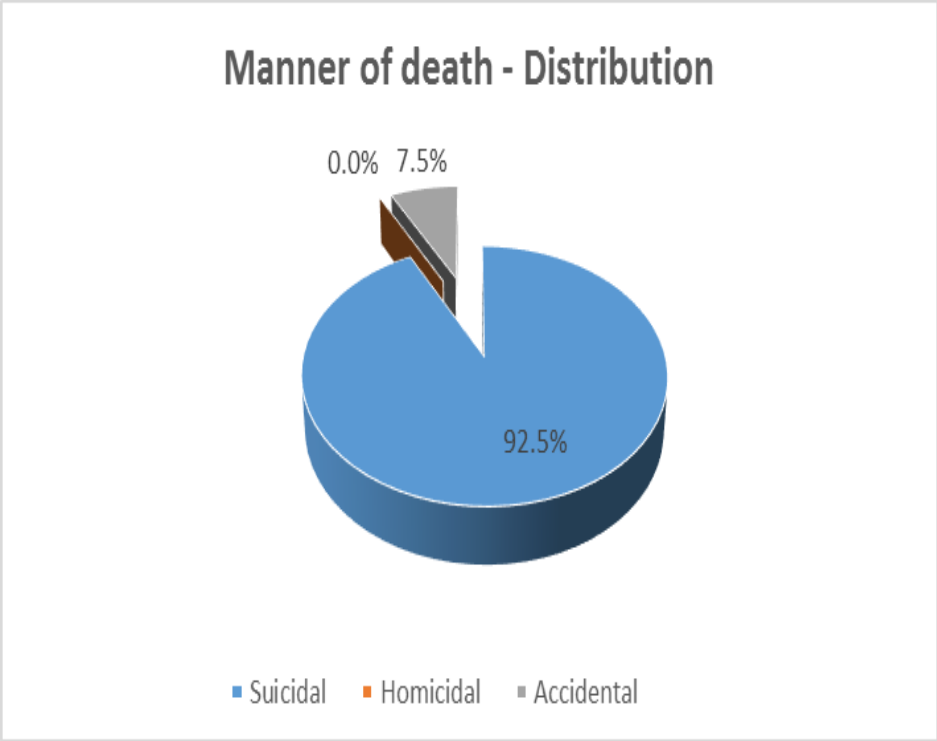


In this study more number of cases time of death was not known (64) 28.2% because many cases were seed dead and brought dead to hospital.

TABLE 17: MANNER OF DEATH

Manner of death	No. of cases	%
Suicidal	**210**	**92.5%**
Homicidal	0	0.0%
Accidental	17	7.5%

FIGURE 17: MANNER OF DEATH

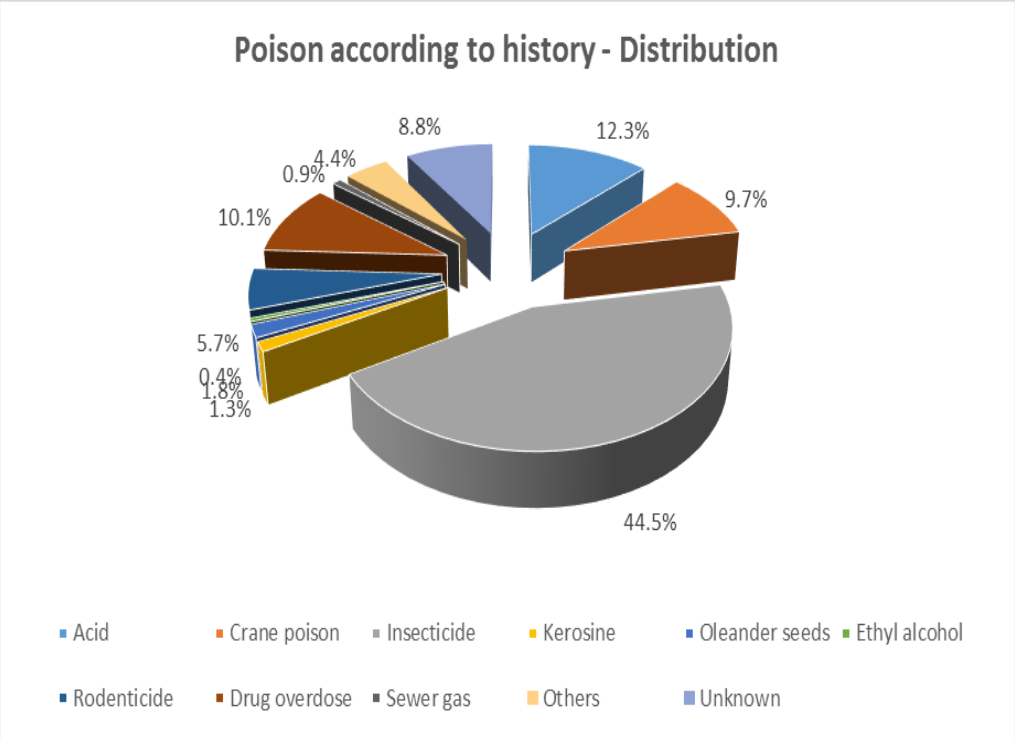


In my study in most of the cases manner of death was sucidal (210) 92.5%

TABLE 18: POISON ACCORDING TO HISTORY

Poison according to history	No. of cases	%
Acid	28	12.3%
Crane poison	22	9.7%
Insecticide	**101**	**44.5%**
Kerosene	3	1.3%
Oleander seeds	4	1.8%
Ethyl alcohol	1	0.4%
Rodenticide	13	5.7%
Drug overdose	23	10.1%
Sewer gas	2	0.9%
Others	10	4.4%
Unknown	20	8.8%

FIGURE18: POISON ACCORDING TO HISTORY



In my study most of the cases consumed insecticide (101) 44.5%

TABLE 19: OTHER POISONS

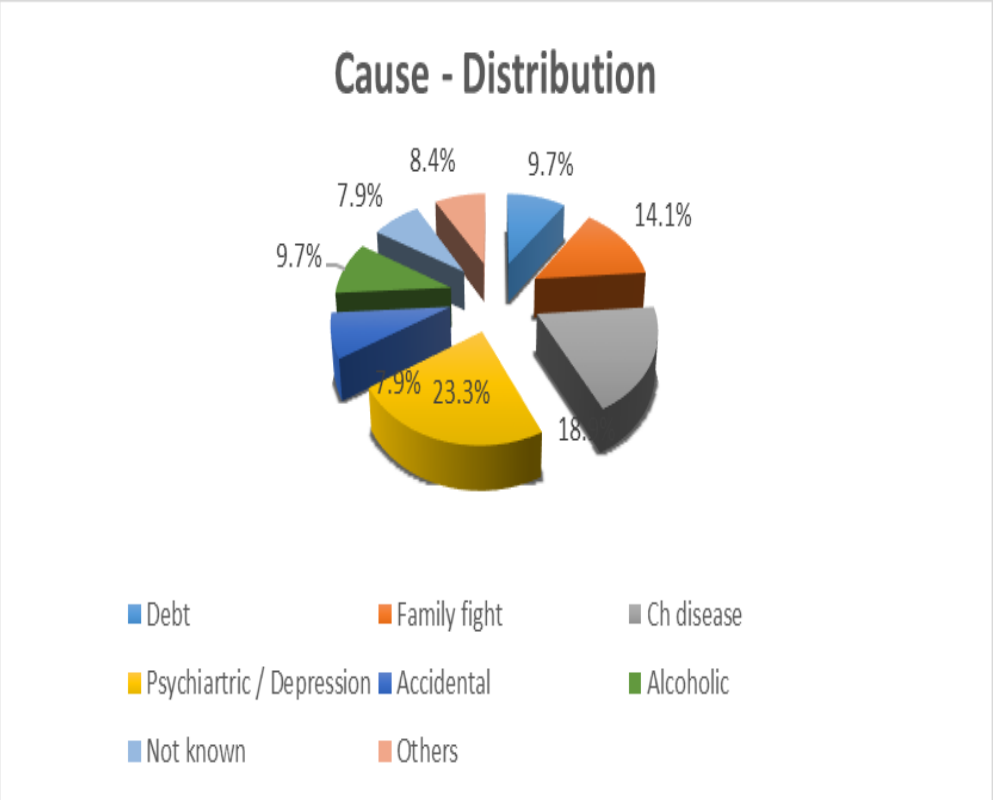
Poison according to history - OTHERS	No. of cases	%
Diesel	1	10.0%
Phenol	3	30.0%
Lyzol	1	10.0%
Machine oil	1	10.0%
Varnish	1	10.0%
Harpic	1	10.0%
Super vasmol	1	10.0%
After shaving lotion	1	10.0%

This table enumerates all the other rare poisons consumed in this study

TABLE 20: CAUSE OF CONSUMPTION OF POISON

Cause	No. of cases	%
Debt	22	9.7%
Family fight	32	14.1%
Ch disease	43	18.9%
Chronic Depression/ Psychiatric	**53**	**23.3%**
Accidental	18	7.9%
Alcoholic	22	9.7%
Not known	18	7.9%
Others	19	8.4%

FIGURE 20: CAUSE OF CONSUMPTION OF POISON

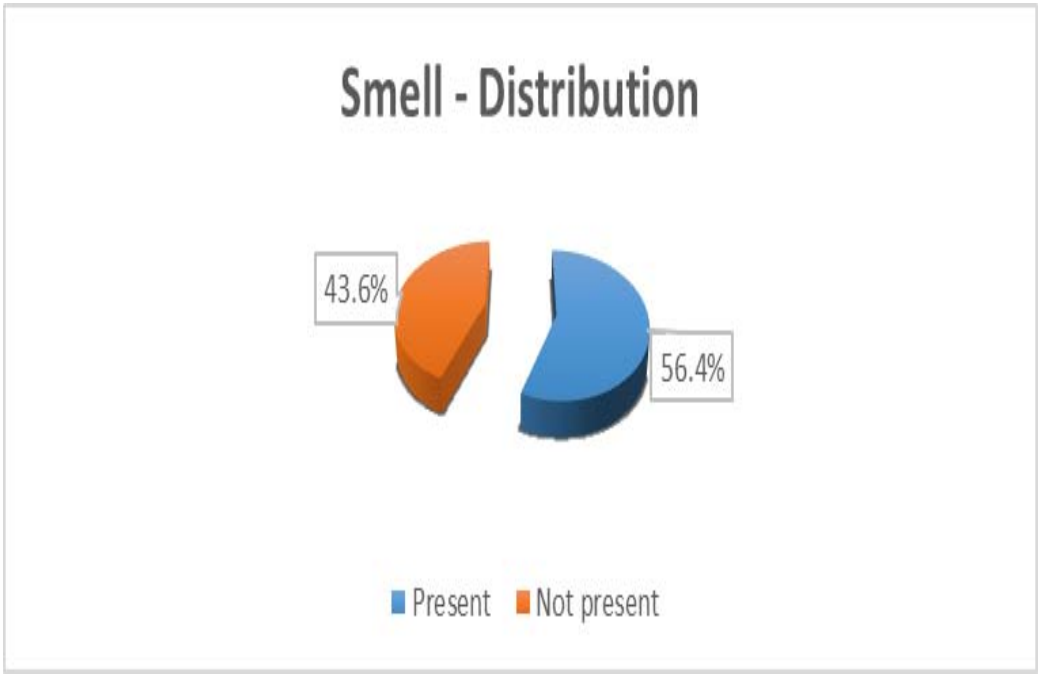


In my study most common reason for consuming poison was chronic depression or psychiatric illness 23.3% (53 cases)

TABLE 21: PRESENCE OF SMELL

Smell	No of cases	%
Present	128	56.4%
Not present	99	43.6%

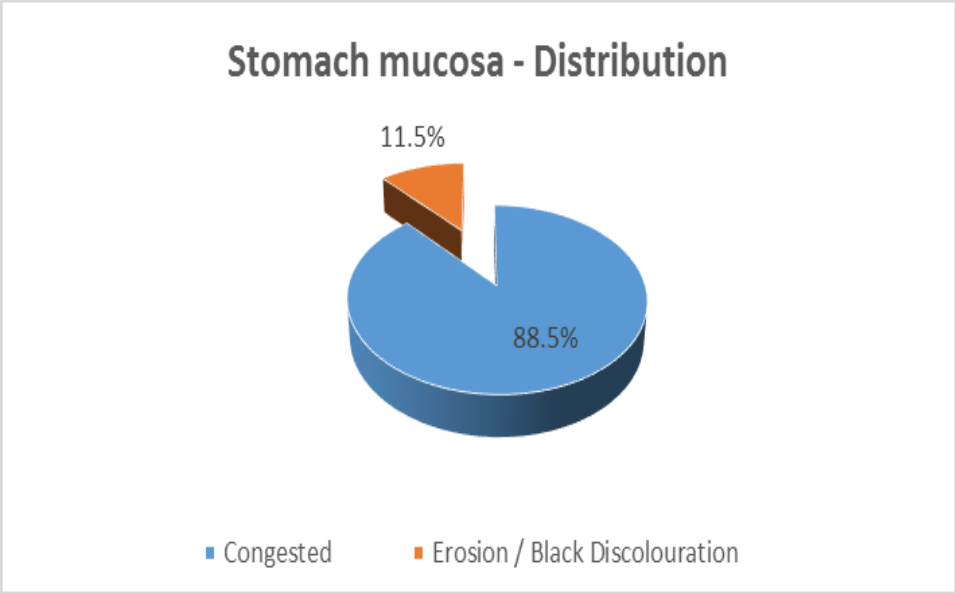
FIGURE 21: PRESENCE OF SMELL



In this study peculiar smell was noted in postmortem examination in 56.4% of cases (128)

TABLE 22: APPEARANCE OF STOMACH MUCOSA

Stomach mucosa	No. of cases	%
Congested	**201**	**88.5%**
Erosion / Black Discoloration	26	11.5%

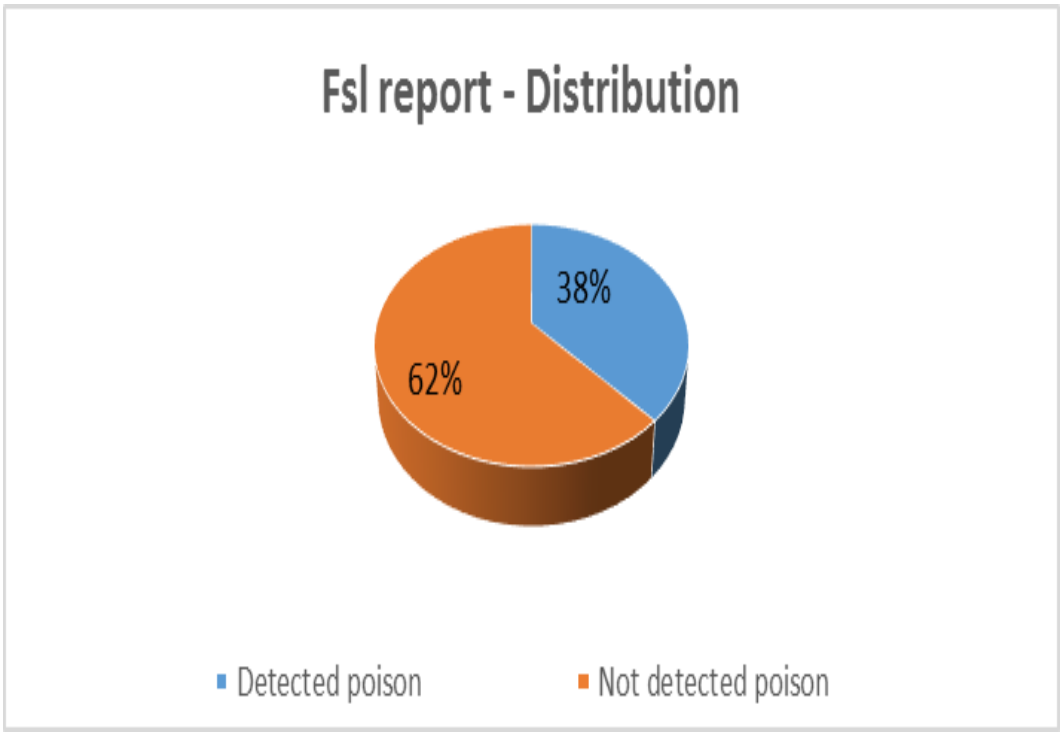


In this study postmortem examination showed congestion of gastric mucosa in 88.8% cases (201)

TABLE 23: FORENSIC SCIENCE LAB REPORT

Fsl report	**No. of cases**	**%**
Detected poison	86	37.9%
Not detected poison	**141**	**62.1%**

FIGURE 23: FORENSIC SCIENCE LAB REPORT

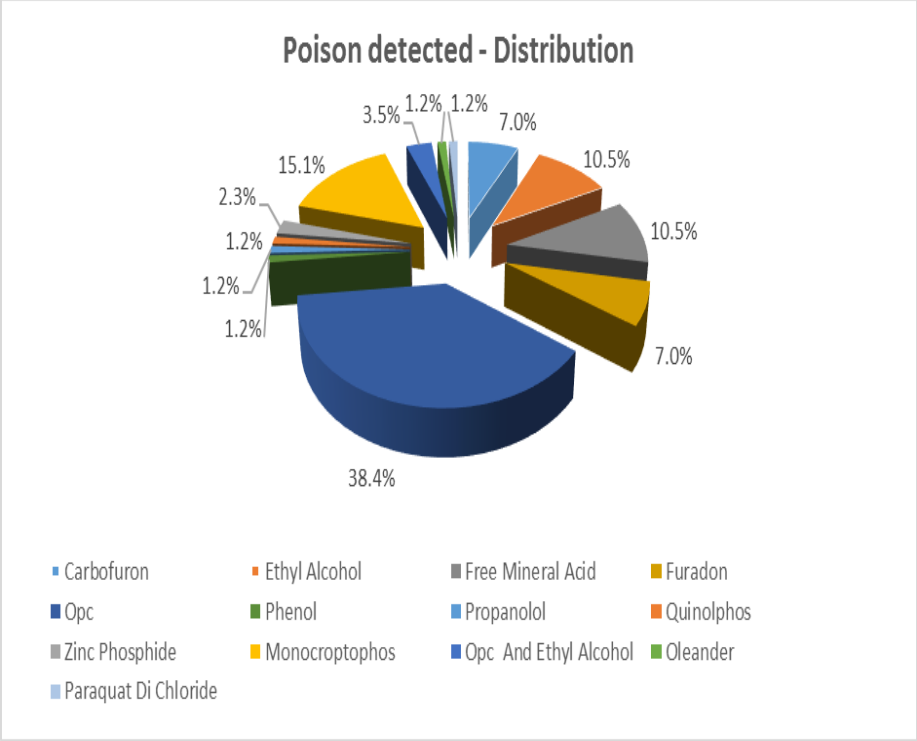


In my study forensic science lab report shows non dedection of poison in maximum cases (141) 62.1%

TABLE 24: POISONS DEDECTED IN FSL

Poison detected	No of cases	%
Carbofuron	6	7.0%
Ethyl Alcohol	9	10.5%
Free Mineral Acid	9	10.5%
Furadon	6	7.0%
Opc	**33**	**38.4%**
Phenol	1	1.2%
Propanolol	1	1.2%
Quinolphos	1	1.2%
Zinc Phosphide	2	2.3%
Monocroptophos	13	15.1%
Opc And Ethyl Alcohol	3	3.5%
Oleander	1	1.2%
Paraquat Di Chloride	1	1.2%

FIGURE 24: POISON DETECTED IN FSL



In this study among the poisons dedected by FSL organophosphorus was the highest (33) 38.4%

DISCUSSION

Self-suicidal poisoning with Pesticide is approximately 1/3rd of all suicides worldwide represents a major hidden public health problem.[58.]

In my study year wise distribution of 227 cases of poisoning reveals that the maximum cases recorded is in the year – 2014 (43.1%), followed by 2013 (31.27%) and 2015 (25.55%). A similar pattern is also noted in [18] where in 1999 - 82 cases of poisoning were seen, which increased to 141 cases in 2002 and then decreased to 118 cases in the year 2003.These types of retrospective studies of poisoning cases were also conducted by [11,25,26] .

In my study, males constituted 70% and females constituted only 30%% of the total victims. The earning member of the family are the males. They undergo the stress. This explains the rising suicidal tendency amongst males. Similar findings was found in the studies conducted by [13,15,16,17,18,20,21,22,25] however the study conducted by, [26] female incidence was found to be more common.

In my study age between 41-50 years was found to be more vulnerable to suicidal poisoning. 41-50years age group undergo lot of stress due to family and financial problems. Our findings were in agreement with the study done by Margaret Warner and Shin.[64,65] Age wise distribution of poisoning cases reveals that the maximum cases were in age group of 20-29 years, in [11,13,15,16,17,18,20,21,22,25] these studies.

In my study month wise distribution of poisoning cases reveals that the incidence is less during summer season, however in the studies conducted by, [11,13,25] revealed that, the incidence in their studies was more common during summer season.

In my study majority of the victims were Hindus consisting of 210 (92.5%) cases. More number of cases 180 (79.3%) were married. Similar findings were noted in the following studies conducted [10,11,15,17,18]. This is due to increased stress level among married both due to marital disharmony and financial instability.

In my study Sex wise marital status year wise reveals poisoning is more common among married people. Married males (40.5%), married females (22.0%). Unmarried males (28.2%) and unmarried females (9.25%) are the least. Study conducted by, **[11]** shows, that poisoning was more common in married men (67.92%), followed by unmarried males (17.92%), married females (12.74%) and unmarried females forms only (1.39%) of poisoning cases. Study conducted by, [18] found that poisoning was more common in married males followed by married females. Studies conducted by, **[7,9,16]** reveals that married individuals are more prone to poisoning when compared to the unmarried.

In my study Socio economic status wise distribution reveals poisoning is more common among lower socioeconomic group (70.5%) followed by middle socio economic group (29.1%%) and least common among high socio economic group (0.4%). Similar studies with similar classification of economic strata i.e. Modified B.G.Prasad's classification was done by,[16] reveals that majority of cases belongs to Class iii and Class iv, followed by class v and vi and least among class i and ii. (57.69%, 32.69% and 9.61%), which is similar to our study (70.5%, 29.1% and 0.4%).Raising demand for daily living leading to

financial needs causes stress and depression among lower socio economic status population. Also due to inability to afford the standard of treatment after exposure.

In my study Occupational distribution shows more cases among daily wage worker 77(33.9%), followed by housewives 47(20.7%). As we received more cases from Chennai, farmers were not leading the list. But the Daily wage workers were more due to low earning and insecurity of job, more among them were alcoholics which is also a prime cause for suicidal poisoning among them. Housewives have no outlet of their emotions due to disharmony of family or no personal money to see their small needs by themselves. In similar studies showed farmers and then housewives[10.] Farmers followed by housewives and then labor[15,] farmers followed by labors [26].

In my study Educational status among the cases, 96 (42.3%) have done their primary education, followed by 69 (30.4%) were illiterate. Less knowledge about the poison, less earning will be the cause of death among them. Similar finding were noted in the following [23] study.

In my study Area wise distribution of poisoning reveals urban population (73.6%) is more than rural population (26.4%). Studies done by, **[11,13,20,21,22]** also reveals that poisoning was more common in the rural than urban area. However studies done by, **[17,25]** shows that poisoning was more common among urban areas. This may be due to that the study was conducted in Chennai.

In my study type of family were mostly nuclear 198 (82.2%) this might be due to no elderly person to guide them or to solve if there is any family disharmony. Similar findings were seen in studies conducted by [23.]

In my study 225(99.1%) of cases consumed poison orally. This is because most of the poison were pesticides and oral intake is the easiest method. In this study 2 cases were due to inhalation of poisonous sewer gas at work place.

In my study 189 (83.3%) cases consumed poison at home, this can be due to availability of poison at home, outburst of emotion in a fight at home or they return home after a tiring and disappointing day. Next to it was 30 (13.2%) consumed poison outside home, like park, inside auto, bridge side, on road, etc.

In my study Time of consumption of poison was found to be 6 PM TO 12 AM 78(34.4%), due to stressful and disappointed day. Similar findings were seen in study conducted by (26) and consumption of poison was noted in day time in [11,13] studies.

In my study Manner of poisoning reveals that 210 (92.5%) of cases were suicidal poisoning followed by accidental poisoning 17 (7.5%). No homicidal poisoning were reported in this study. Studies conducted by, **[17,18,22]** also reveal that suicidal poisoning is more common than homicidal poisoning. In the study conducted by, [22] also, not a single case of homicidal poisoning was noted. In the study conducted by, [15,26] also revealed intentional poisoning was more common than the accidental poisoning.

In my study It was also found that 45 (19.8%) were brought dead and 27(11.9%) were seen dead, however the maximum number of cases succumbed

to death on treatment after 24hrs. In (20) study shows 18.6% died during admission. Amongst the 227 victims of poisoning, 150 (66.1%) cases undergone treatment.

In my study Chronic Depression was the main reason for poison consumption in 53 (23.3%) of the deaths. Chronic depression due to alcoholism, unemployment, psychiatric or illness, family disharmony and so on. Similar findings were seen in the following study [15] .

In this study, we observed that Insecticides was the most common consumed poison which accounted for 101 (44.5%). Seen in the following study [30,38,43] Followed by acid consumption 28 (12.3%) and the unknown drug overdose 23 (10.1%). Different type of poisons seen in this study are diesel, varnish, super vasmol, after shaving oil and machine oil.

In my study During Postmortem peculiar smell was noted in 128 (56.4%) , Gastric mucosa was congested in 201 (88.5%), black discoloration and erosion of gastric mucosa was seen in 26 (11.5%).

In my study Forensic science laboratory detected Poison in 86(37.9%) out of it organophosphorus was the maximum 33(38.4%). Followed by 13(15.1%) monocroptophos and then 9 (10.5%) cases each ethyl alcohol and free mineral acid.

SUMMARY & CONCLUSION

A retrospective study of deaths due to poisoning among the medico legal autopsy conducted in the mortuary of Government Kilpauk Medical College Hospital, Chennai-10, during the period b0etween January 2013 to December 2015. The present study revealed the following:

1. 9031 cases were brought for post-mortem examination out of which 227 (2.5%) deaths were due to poisoning.
2. Out of 227 autopsied poisoning victims, 92.5% of cases were suicidal poisoning.
3. There were 159 (70.0%) male and 68 (30.0%) female amongst poisoning cases.
4. In this study maximum number of victims 68 (30%) belonged to age group of 41-50 years.
5. Most of cases 189 (83.3%) took place at home and 160 (70.5%) victims belonged to lower socioeconomic status.
6. Majority of the victims were belonging to Hindu consisting of 210 (92.5%) cases.
7. 180 (79.3%) were married. 40% of the males were married.
8. In our study, most of cases 78 (34.4%) consumed poison during 6pm to 12am.
9. A greater number of suicidal poisoning cases 81 (35.7%) were in the summer.

10. It was also found that 45 (19.8%) were brought dead and 27(11.9%) were seen dead.
11. The maximum number of cases succumbed to death on treatment after 24hrs.
12. Amongst the 227 victims of poisoning, 150 (66.1%) cases undergone treatment.
13. Chronic Depression was the main reason for poison consumption in 53 (23.3%) of the deaths.
14. In this study, we observed that Insecticides was the most common consumed poison which accounted for 101 (44.5%).
15. Forensic science laboratory detected Poison in 86(37.9%)
16. Organophosphorus was the maximum 33(38.4%) in the detected cases.

RECOMMENDATIONS

Following measures are highly recommended to reduce deaths due to Poisoning:

1. Government should implement strict rules to decrease the easy accessibility and availability of pesticides.
2. Health education to adolescents at school and college level about poisoning and its first aid treatment should be done.
3. House wives should get involved in recreational activities. Also strict implementation of anti-dowry law, marriage counseling and women empowerment should be done.
4. Persons with psychosocial problems should be identified at the earliest and should be referred for psychiatric counseling. Educate the family to support them in recovering.
5. To avoid accidental domestic poisoning all domestic poisoning must contain the caption “POISON” and to be advised to keep safely out of reach from children.
6. Poison information center should be initiated in each district throughout the country. It helps in timely diagnosis and treatment.
7. All the hospitals should have separate toxicological unit exclusively dealing with clinical poisoning cases.
8. Improve the facilities in Primary Health Center to treat all poisoning causalities.

9. Educating NGO's, village head and other volunteers about the first aid treatment of poisoning at household level is a good way to treat causality immediately.
10. Further research is needed to introduce safer pesticides with minimal harm to the human.
11. Proper preservation of viscera and packing to be done after postmortem.
12. Detailed history and specific clues like empty tablet strips taken by the deceased should be collected from the relatives of the deceased.
13. All these information should be conveyed to the FSL toxicology department along with request from, it will help them to narrow down to a group of poison.

BIBLIOGRAPHY

1. Modi NJ. Textbook of Medical Jurisprudence & Toxicology. 22nd Edn. Bombay; Butterworth. 1999: 230-240.
2. Narayan Reddy KS. Essentials of Forensic Medicine & Toxicology. 26th edn. Hyderabad: K.Sugunadevi. 2007: 278-293.
3. Santhosh CS, Kumar S, Nawaz B. Profile of poisoning cases autopsied at district government hospital, Davangere. IJFMT 2012Jan-June;6(1):104-6.
4. Pillay.V.V. Modern Medical Toxicology, (Completely Updated Revised and Profusely Ilustrated), 4th Edition, Delhi, Jaypee Brothers Medical Publishers Pvt., Ltd., 2013.
5. Dr.Vinesh Shah.B changing trends of poisoning in Surat city, Ph.D Thesis (A Thesis submitted to Veer Narmed South Gujarath University, November 2007)
6. Crime in India 2014.National crime records bureau. Ministry of home affairs statistics. URL:http://ncrb.nic.in/ADSI2014/adsi-2014%20full%20report.pdf
7. Krishnan Vij, Text Book of Forensic Medicine and Toxicology Principles & Practice, 5th Edition, New Delhi, Reed Elsevier, India Pvt., Ltd., 2011.

8. Dr.Vinesh Shah.B changing trends of poisoning in Surat city, Ph.D Thesis (A Thesis submitted to Veer Narmed South Gujarath University, November 2007)
9. Rajesh Bardale, Principles of Forensic Medicine and Toxicology, 1st edition, Delhi, Jaypee Brothers Medical Publishers Pvt., Ltd., 2011.
10. Death due to Poisoning - a Medicolegal Study at Dhaka Medical College, Dhaka. Mushtaq Ahmad, Farial Naima Rahman, Md Mozaharul Islam, Md Rakib Uddin Majumder
11. Bharath K Guntheti Udaypal Singh, "The Pattern of Poisoning in khammam" J.Indian Academy of Forensic Medicine, October – December 2011, Vol 33, No.4 Page – 296-300.
12. A hospital based epidemiological study of deaths due to organophosphorus compound poisoning was conducted by S Peranantham1, Kusa Kumar Shaha1, Ajit Sahai2, Siddhartha Das1, G Manigandan1, K Shanmugam
13. Shreemanta Kumar Dash, Manoj Kumar Mohanthy, Kiran Kumar Patnaik, Sachidananda Mohanthy, "Socio Demographic Profile of Poisoning Cases" JIAFM, 2005, 27 (3). ISSN 0971-0973, Page – 133-138
14. Toxiepidimiology of acute poisoning: the trend of poisoning cases admitted to the Government District Headquarters Hospital, conducted by Indu TH, Raja D, Ponnusankar S

15. Ramanath K.V. Naveen Kumar H.D., “Study the assessment of poisoning cases in rural Tertiary care teaching Hopsital by clinical Pharmacist”. Asian Journal of Pharmaceutical and Clinical Research. ISSN-0974-2661, Vol 5, Issue 2, 2012
16. Shoaib Zaheer.M, Aslam.M, Vibanshu Gupta, Vibhor Sharma and Shadab Ahmad Khan, “Profile of Poisoning cases at a north Indian tertiary care hospital”. Health and Population: Perspectives and issues Vol.3214, 176-183, 2009, Page no-176-183.
17. Dr. Gargi.J, Dr. Hakumat Rai, Dr. Ashok Chanana, Dr.Gurmanjit Rai etal “Current trends of Poisoning – A Hospital profile”..Journal of Punjab Academy of Forensic Medicine & Toxicology, 2008, Page no – 38-42.
18. Unnikrishnan B, Singh B, Rajeev A, “Trends of acute poisoning in south Karnataka.” Kathmandu University Medical Journal. Vol.3, No.2, Issue 10, 2005, Page No-140-154.
19. A retrospective study of poisoning cases in Melmaruvathur region of Tamil Nadu for period of 3 years, by O. Gambhir Singh, A. M. Singh , IAIM, 2014; 1(4): 27-31
20. Tejas Prajapati, Kartik Prajapati, Rakesh Tandon, Saumil Merchant “Acute Chemical and Pharmaceutical Poisoning cases treated in civil Hospital, Ahmedabad: One year study Asia Pacific Journal of Medical Toxicology APJMT 2;2, June – 2013, Page – 63-67.

21. Subash Vijaya Kumar, Venkateswarlu.B, Sasikala.M, and Vijay Kumar.G “A Study on Poisoning Cases in a tertiary care hospital” Indian Journal of critical care Medicine, IJCCM – 2009, Page No- 33-39.
22. Vinay B shetty. Gurudatta S Pawar., Inamadar. P.I. “Profile of poisoning cases in district and medical college hospitals of north Karnataka”. Indian Journal of Forensic Medicine & Toxicology Vol.2, No.2, July-December – 2008, Page No-26-28.
23. Profile of suicidal poisoning in Puducherry area conducted by Naveen N, Madhuvardhana T, Arun M, Balakrishna Rao A J, kagne R N.
24. “Use it to Dye not to Die” A study conducted at Sri Venkateswara Institute of medical sciences (SVIMS University) Tirupati – Andhra Pradesh. Shows hair dye as the commonest emerging suicidal poison in the year – 2013, The Hindu, 25-10-2013, Friday, Page – 2 (Region) www.thehindu.com.
25. Ansam F Sawalha., Waleed M Sweileh., Maysoon T Tufaha. and Dua Y Al-jabi.. “Analysis of the pattern of acute poisoning in patients admitted to a government hospital in palestine” Bari & Clinical Pharmacological society, 2010, page no-914-918.
26. Deepak Pokhrel, Sirjana pant, Anupama Pradhan, Saffar Mansoor “A comparative Retrospective study of poisoning cases in central, zonal and District Hospitals”, Kathmandu University Journal of Science,

Engineering and Technology, Volume-1, No-v, September – 2008, Page No-40-48.

27. Sanjay Gupta, Kumar. S ,Sheikh M.I. “Comparitive study and changing trends of poisoning in the year 2004-2005, at Surat, India, International Journal of Medical Toxicology & Legal Medicine Vol.10,No.1, July-December-2007, Page No.16-20
28. Dr.Kartik Prajapathi, Dr.Saumil Merchant.P, Dr.Pratik Patel.R “Trends of Suicidal Poisoning in Ahmedabad (Retrospective study)” NHL Journal of Medical Sciences, Volume.1, Issue 1, July-2012. Page.No.18-22.
29. Navin Kumar M, Varma, Kalele S.D. “Study of Profile of Deaths due to poisoning in Bhavnagar region”, Journal of Indian Academy of Forensic Medicine, Volume-33, No-4, ISSN 0971-0973 October-December-2011.
30. Manoj Kumar Mohanty, Arun M, Ritesh Menezes.G, Vikram Palimar, “Correlation between postmortem diagnosis and survival time in poisoning Deaths”. Journal of Indian Academy of Forensic Medicine 27(1) ISSN.0971-0973, 2005, Page.no.23-27.
31. Kartik Prajapathi, Saumil Merchant P, Pratik Patel R “Trends of Suicidal poisoning in Ahmedabad – A Retrospective study”, Indian Journal of Forensic Medicine and Toxicology, Volume-7, No-1, January – June, 2013, Page.No.82-86.

32. Gupta, B.D, Vaghela P.C. “Profile of Total Poisoning in and around Jamnagar”, Journal of Indian Academy of Forensic Medicine 27(3). Iss No.971-0973, 2005, Page No.145-148.
33. Kishan Siddhapur. R,Gurudatta Pawar.S, and Shashidhar C Mestri “Trends of Poisoning and Gross Stomach Mucosal appearance in Fatal poisoning cases. An autopsy study”. Journal of Indian Academy of Forensic Medicine, Volume.33, No-2, April-June-2011, ISSN.0971-0973, Page.No.106-111.
34. Cyriac Job, “A Retrospective study of poisoning cases in Thrissur District of Kerala for the year 1995, Journal of the Indian Society of Toxicology (JIST), Volume 005, Issue 001, January-June-2008, Page.No.23-27.
35. Dr.Karamjit Singh, Dr.Oberoi, S.S., Dr. Bhullar.D.S. “Poisoning trends in the malwa region of Punjab” Punjab academy of forensic Medicine & toxicology year – 2007 , page- 41-44.
36. Somasundaram K.V., Ashok patil, Shukla.S.K. “Epidemiological profile of poisoning cases treated at pravara hospital, Loni, India” Indian Journal of Preventive Social Medicine Volume.40, No.3 & 4 July-December, 2009, Page.No.184-188.
37. Amit Patil.M., Satin Meshram.K., Rajesh Kharat.D, Mohite.S.C etal “Profile of fatal methyl Alcohol poisoning outbreak -A Medico legal Autopsy Case Study”. Indian Journal of Forensic Medicine & Toxicology, Volume.7, No.1, January-June-2013, Page.No.16-20.

38. Kora S.A., Doddamani G.B., Halagali G.R, Vijayamahantesh. S.N etal.“Socio Demographic profile of organophosphorous poisoning cases in southern India” Journal of clinical and diagnostic Research, Volume 5(5) October-2011, Page.No.953-956.
39. Selvam.V., Panner Selvam.G., Vijayanath.V., “Study of Death Incidence by Inscecticide poisoning in Salem” International Journal of Medical Toxicology and Forensic Medicine, Page.No.20-26.
40. Omid Mehrpour, Mandana Dolati, Kambiz soltaninejad, Shahin Shadnia, Bashir Nazparvar. “Evaluation of histo pathological changes in fatal aluminium phosphide poisoning, Indian Journal of Forensic Medicine & Toxicology, Volume.2, No.2, July-December-2008, Page.No.34-36.
41. Gannur D.G., Prakash Maka, Narayana Reddy K.S., “Organophosphorous compound poisoning in Gulbarga region - A five year study: Indian Journal of Forensic Medicine and Toxicology, Volume-2, No.1, January-June-2008, Page.No.3-11.
42. Dr.Zariwala.R.C., Dr.Bhise.R.S., Dr.Patel D.S Dr. Solanki A.M, etal “A Retrospective study of 5 years organophosphorous poisoning in Ahmedabad.” Journal of Indian Academy of Forensic Medicine, August-2011, Page.No.18-22.
43. Dhaval Patel.J, Pawan Tekade.R. “Profile of Organophosphorous poisoning at Maharani Hospital, Jagdalpur, Chattisgarh. A three years

study" Journal of Indian Academy of Forensic Medicine, Volume.33, No.2, April-June-2011, ISSN.0971-0973, Page.No.102-105.

44. Hareesh kumar R.S, Raju.K, Chandan.V, Sidramappa Gouda, Rashmi Belodu, Patil SB, Patil BS. "Epidemiology and Clinical study of snake bite cases admitted in basaveshwara Hospital, Chitradurga, Karnataka, Indian Journal of Forensic Medicine & Toxicology, Vol – 7, No -1, January – June – 2013, Page No – 58-60.
45. Dr.Kar.S.M, Dr.Siddhartha Timisinha, Dr.Prasanth Agarwal "An Epidemiological study of organphosphorus poisoning at Manipal Teaching Hospital, Pokhara, Nepal." Journal of Indian Academy of Forensic Medicine 32 (2), ISSN.0971-0973, Page.No.108-109.
46. Thakur M.S, Naik J.D, Sheetu jailkhani MK, Langare SP "A One year retrospective study of snake bite cases admitted in a tertiary care hospital of western Maharashtra" International journal of Biology & Medical Research 2013 4(2) : 3115 – 3117.
47. Ashok Kumar Jain, Dubey B.P., Garg S.P, Manish Nigam. "Trends of Aluminium phosphide poisoning in Bhopal region – A retrospective study of 10 years". Journal of Indian Academy of Forensic Medicine, 31(3), Page.No.234-236.
48. Dr.Sanjeev Kumar Chowdhary, Dr.Momin S.G., Dr.Dipak Vora.H, Dr.Pikesh Modi etal., "An Epidemiological Study of Fatal Aluminium phosphide poisoning at Rajkot" IOSR Journal of Pharmacy, Volume-3, Issue-1, January-Feburary-2013, Page.No.17-23

49. Ananda Kumar L, Sai Prasad B.V., Sobha Rani.G, Mohammad Faheem. K “Hairdye – An Emerging Icon – As a Suicidal agent – A short report.” Indian academy of Forensic Medicine, 2012, Page- 33-36.
50. Bhargava.P, Matthew.P “Hair Dye Poisoning” JAPI.Volume55, December-2007, Page No-871-872.
51. Uzma Saleem, Saeed Mahmood, Bilal Ahmad, Alia Erum, Saira Azhar, Bashir Ahmad. “Benzodiazepines poisoning cases, a retrospective study from Faisalabad, Pakistan”. Pakistan Journal of Pharmacy 20-23 (1&2) 2007-2010, Page.No.58-60.
52. Pratik Vijay Tarvadi, Shankar Bakkanavar. M, Manjunath.S “Trends of Poisoning among children at Kasturba Hospital, Manipal, Nitte University Journal of Health Science (NUJHS, Volume-3, No-2, June-2013, ISSN 2249-7110) Page No.25-28.
53. Surjit Singh, Sunil Singhi, Sood.N.K “Changing Pattern of Childhood poisoning (1970-1989) Experience of a large north Indian Hospital, Indian Pediatrics Journal Volume-32, March 1995, Page.No.331-336.
54. Vikram Palimar and Pradeep Kumar.G “Poisoning deaths in children”, Journal of Indian Academy of Forensic Medicine, 31 (3) Page.No.218-222.
55. Gupta-S, Govil.Y.C., Misra.P.K., Nath.R, Srivastava K.L., “Trends in poisoning in children: Experience at a large referral teaching hospital”. The National Medical Journal of India, Volume-11, No.4, 1998, Page.No.166-168.

56. Brojen Singh T, Romeo Singh.K, Lallan Prasad Taruni N.G, Bijoy Sing T.H “Child hood poisoning – A 3 years experience in RIMS, Imphal”. Journal of Forensic Medicine and Toxicology, Volume-26, No:1 - January-June-2009, Page-No-37-41.
57. Nowneet kumar Bhat, Minakshi Dhar, Sohaib Ahmad, Vipan Chandar “Profile of Poisoning in children and adolescents at a north Indian Tertiary care center” Journal, Indian Academy of Clinical Medicine Volume 13, No-1, January –March-2012, Page-No-37-42.
58. Bertolote JM, Fleischmann A, Eddleston M, Gunnell D. Deaths from pesticide poisoning: a global response. British J Psych 2006;189:201-203. Available at: http://bjp.rcpsych.org/content/ 189/3/201.short Accessed Jan 30, 2014
59. S P Singh et al. Study of poisoning trends in north India - A perspective in relation to world statistics. Journal of Forensic and Legal Medicine 2013; 20: 14-18.
60. Mizanur Rahman, Habibuzzaman Chowdhury et al. Study of poisoning-the most prevailing cause of unnatural death in North Bengal. JSSM Jun 2010, 2(1): 4-5.
61. Bharath K. Guntheti, Uday Pal Singh. The Pattern of Poisoning in Khammam. JIAFM October- December 2011; 33(4):296-300.
62. Mrinal Haloi, Mamata Devi Haloi, Amarjyoti Patowary. Death due to Poisoning in District of Kamrup, Assam -A Medico-legal Study. JIAFM Jan-March 2013; 35(1):17-20.

63. Margaret Warner. Drug Poisoning Deaths in the United States, 1980–2008. NCHS Data Brief; No. 81; December 2011.

64. Shin SD, Suh GJ, Rhee JE, Sung J, Kim J. Epidemiologic characteristics of death by poisoning in 1991-2001 in Korea. J Korean Med Sci. Apr 2004; 19(2):18-94.

ANNEXURE I:

PROFORMA:

S.NO	QUESTION	DETAILS						
1	Pm no							
2	Age							
3	Sex	Male				Female		
4	Religion	Hindu			Christian		Muslim	
5	Educational status	ILLITRATE	Primary School	Middle school	High school	diplomo	graduate	Professional
6	Occupation	unemployed student house wife daily wage worker professional business farmer other jobs						
7	Marital status	Married			Unmarried		Divorced	
8	Type of family	Nuclear			Joint		Unknown	
9	Socio economic status	Lower		Middle		High		Unknown
10	Domiciliary	Rural				Urban		

12	Season	Summer	Rainy	autumn	Winter
13	Route of exposure	Oral	IV	IM	Others
14	Place of consumption	Home	Workplace	Outside	Others
15	Time of consumption	6am-12pm 12pm-6pm 6pm-12am 12am-6am not known			
16	Treatment	Done		Not done	
17	Duration of survival	seen dead Brought Dead <6h 6-12h 12-24h >24h			
18	Time of death	not known 6am-12pm 12pm-6pm 6pm-12am 12am-6am			
19	Manner of death	Succidal	Homicidal	Accidental	
20	Poison according to history				
21	Cause	debt family fight chronic disease psychiartric/ depression accidental alcoholic not known others			
23	Peculiar Smell	Present	Not present		
25	Stomach mucosa	Congested	Eroded /blackening/perforation		
28	Fsl report				

ANNEXURE – II

ABBREVIATIONS USED IN MASTER CHART

AGE DISTRIBUTION:

0-10	-	0-10 years
11-20	-	11-20 years
21-30	-	21-30 years
31-40	-	31-40 years
41-50	-	41-50 years
51-60	-	51-60 years
61-70	-	61-70 years
>70	-	more than 70 years

SEX

M	-	Male
F	-	Female

RELIGION:

H	-	Hindu
M	-	Muslims
C	-	Christians
O	-	Others

OCCUPATION:

H.W	-	House wife
UNE	-	unemployed
DWW	-	Daily wage worker
JOB	-	Business
PR	-	Professional
ST	-	Student
FR	-	Farmer
OT	-	Other jobs

MARITAL STATUS:

MR	-	married
UM	-	unmarried

SOCIO-ECONOMICAL STATUS:

UPPER	-	Upper
MIDDLE	-	Middle
LOW	-	Lower

DOMICILIARY:

URBAN	-	Urban area
RURAL	-	Rural area

EDUCATIONAL STATUS:

PROF	-	Professional
GRADUATE	-	Graduate

HIGH	-	High School
MIDDLE	-	Middle School
PRIMARY	-	Primary School
ILITERATE	-	Illiterate

PLACE OF INCIDENT:

HOME	-	Home
W.P	-	Working Place
OUT SIDE	-	Out side

SEASONAL VARIATION:

S	-	Summer
R	-	Rainy
W	-	Winter
A	-	Autumn

TIME OF INCIDENT:

6A- 12P	-	6AM TO 12PM
12P- 6P	-	12PM TO 6PM
6P- 12A	-	6PM TO 12AM
12A-6 A	-	12 AM TO 6 AM
UNK	-	UNKNOWN

MANNER OF DEATH:

AC	-	Accidental
SU	-	Suicidal
HO	-	Homicidal

PERIOD OF SURVIVAL:

BD	-	Brought dead
SD	-	seen dead
<6 HRS	-	Up to 6 hours
6-12 HRS	-	6 to 12 hours
12-24 HRS	-	12 to 24 hours
>24hrs	-	more than 24hours

TIME OF DEATH:

6A- 12P	-	6AM TO 12PM
12P- 6P	-	12PM TO 6PM
6P- 12A	-	6PM TO 12AM
12A-6 A	-	12 AM TO 6 AM
UNK	-	UNKOWN

TREATMENT

D	-	DONE
ND	-	NOT DONE

REASON FOR CONSUMPTION OF POISON

D	-	Debt
FF	-	Family fight
CD	-	Chronic disease
PD	-	Psychiatric / depression
A	-	Accidental
AL	-	Alcoholic
NK	-	Not known
O	-	Others

PECULIAR SMELL

P	-	PRESENT
NT	-	NOT PRESENT

GASTRIC MUCOSA

C	-	Congested
E/B	-	Eroded/ blackish discoloration

FSL DEDECTED

DT	-	detected
NDT	-	Not Detected

POISONS

OPC	-	Organophosphorus
EA	-	Ethyl alcohol
FMA	-	Free mineral acid
P	-	Pesticide
R	-	Rodenticide
I	-	Insecticide
SG	-	Sewer gas
DO	-	Drug Overdose
OS	-	Oleander seed
K	-	Kerosene
CP	-	Crane poison
M	-	Monocroptophos
C	-	Carbofuron
F	-	Furadon
PH	-	Phenol
PRO	-	Propanalol
Q	-	Quinolphos
ZP	-	Zinc phosphide
PDC	-	Para Di Chloride

ANNEXURE – III

MASTER CHART

Sl. No.	SUBJECT ID	AGE DISTRIBUTION (years)	SEX	RELIGION	OCCUPATION	MARITAL STATUS	SOCIO-ECONOMIC STATUS	DOMICILIARY	EDUCATIONAL STATUS:	PLACE	SEASON	TIME OF INCIDENT	MANNER	PERIOD OF SURVIVAL	TIME OF DEATH	TREATMENT DONE	REASON	PECULIAR SMELL	GASTRIC MUCOSA	FSL DEDECTED	POISONS
1	1	0-10	M	H	UNE	UM	LOW	URBAN	ILITERATE	HOME	S	12P-6P	AC	>24	6A-12A	D	A	NT	C	NDT	
2	2	31-40	M	H	DWW	MR	LOW	URBAN	PRIMARY	HOME	S	6P-12A	SU	>24	12P-6P	D	PD	P	C	NDT	
3	3	41-50	M	H	DWW	MR	LOW	URBAN	ILITERATE	HOME	S	6P-12A	SU	12-24H	12A-6A	D	D	P	C	NDT	
4	4	31-40	M	H	UNE	UM	LOW	URBAN	PRIMARY	HOME	R	6P-12A	SU	>24	12A-6A	D	PD	NT	E/B	DT	FMA
5	5	21-30	M	H	DWW	MR	LOW	URBAN	PRIMARY	HOME	R	12A-6A	SU	SD	UNK	ND	PD	P	C	DT	Q
6	6	>70	F	H	H.W	MR	LOW	URBAN	ILITERATE	HOME	R	6A-12P	SU	<6H	6A-12P	D	CD	NT	E/B	DT	FMA
7	7	41-50	M	H	FR	MR	LOW	URBAN	PRIMARY	HOME	R	12P-6P	SU	6-12H	6A-12A	D	PD	NT	E/B	DT	FMA
8	8	31-40	M	H	OT	MR	LOW	URBAN	PRIMARY	HOME	R	6P-12A	SU	6-12H	6A-12P	D	CD	NT	C	NDT	
9	9	21-30	F	H	H.W	MR	LOW	RURAL	PRIMARY	HOME	R	6A-12P	SU	6-12H	6A-12A	D	PD	P	C	NDT	
10	10	21-30	M	H	DWW	MR	LOW	URBAN	PRIMARY	HOME	R	12P-6P	SU	12-24H	12P-6P	D	D	NT	E/B	NDT	
11	11	31-40	F	H	DWW	MR	LOW	URBAN	PRIMARY	HOME	R	6A-12P	SU	6-12H	12P-6P	D	AL	NT	C	NDT	
12	12	31-40	M	H	FR	MR	LOW	RURAL	PRIMARY	HOME	S	6P-12A	SU	>24	12A-6A	D	PD	P	C	DT	OPC
13	13	31-40	M	C	PR	UM	MIDDLE	URBAN	GRADUATE	HOME	S	UNK	SU	SD	UNK	ND	NK	NT	C	NDT	
14	14	61-70	M	H	JOB	MR	MIDDLE	URBAN	HIGH	OUTSIDE	S	6P-12A	SU	<6H	12A-6A	D	PD	NT	C	DT	OPC
15	15	31-40	M	H	DWW	MR	MIDDLE	URBAN	HIGH	OUTSIDE	S	6P-12A	SU	<6H	6A-12A	ND	PD	P	C	NDT	
16	16	31-40	M	H	PR	MR	MIDDLE	URBAN	GRADUATE	OUTSIDE	S	6P-12A	SU	BD	UNK	ND	NK	P	C	NDT	
17	17	10-19	F	H	H.W	MR	MIDDLE	URBAN	HIGH	HOME	S	6A-12P	SU	BD	UNK	ND	FF	P	C	DT	C
18	18	31-40	M	H	OT	MR	LOW	RURAL	ILITERATE	OUTSIDE	S	12P-6P	SU	<6H	6A-12A	D	AL	P	C	DT	EA
19	19	21-30	M	H	DWW	MR	MIDDLE	URBAN	PRIMARY	HOME	S	12A-6A	SU	>24	12P-6P	ND	NK	P	C	NDT	
20	20	21-30	M	H	UNE	UM	MIDDLE	URBAN	GRADUATE	HOME	S	UNK	SU	SD	UNK	ND	PD	P	C	DT	PRO

Sl. No.	SUBJECT ID	AGE DISTRIBUTION (years)	SEX	RELIGION	OCCUPATION	MARITAL STATUS	SOCIO-ECONOMIC STATUS	DOMICILIARY	EDUCATIONAL STATUS:	PLACE	SEASON	TIME OF INCIDENT	MANNER	PERIOD OF SURVIVAL	TIME OF DEATH	TREATMENT DONE	REASON	PECULIAR SMELL	GASTRIC MUCOSA	FSL DEDECTED	POISONS
21	21	31-40	M	H	DWW	UM	LOW	URBAN	ILITERATE	HOME	S	12P-6P	SU	6-12H	6A-12P	D	AL	P	C	NDT	
22	22	31-40	M	H	FR	MR	LOW	URBAN	PRIMARY	HOME	S	12P-6P	SU	>24	12P-6P	D	D	P	C	DT	OPC
23	23	21-30	M	H	ST	MR	LOW	RURAL	GRADUATE	HOME	W	6P-12A	SU	>24	6A-12P	D	O	P	C	DT	OPC
24	24	21-30	F	H	ST	UM	MIDDLE	URBAN	GRADUATE	HOME	W	12P-6P	SU	SD	UNK	ND	O	P	C	NDT	
25	25	10-19	M	H	DWW	UM	LOW	URBAN	PRIMARY	OUTSIDE	W	6A-12P	SU	BD	UNK	D	O	P	C	NDT	
26	26	21-30	F	H	DWW	MR	LOW	URBAN	ILITERATE	W.P	W	6P-12A	SU	>24	12P-6P	D	CD	P	C	NDT	
27	27	31-40	M	H	OT	MR	LOW	URBAN	ILITERATE	HOME	W	6P-12A	SU	BD	UNK	ND	PD	P	C	NDT	
28	28	41-50	M	H	DWW	MR	LOW	URBAN	PRIMARY	HOME	W	6P-12A	SU	>24	12A-6A	D	AL	P	C	NDT	
29	29	51-60	F	H	H.W	MR	LOW	URBAN	ILITERATE	HOME	W	6P-12A	SU	>24	12A-6A	D	PD	P	C	NDT	
30	30	31-40	M	H	DWW	MR	LOW	URBAN	ILITERATE	HOME	W	12P-6P	SU	BD	UNK	ND	AL	NT	C	DT	OPC
31	31	51-60	M	H	DWW	MR	LOW	URBAN	ILITERATE	HOME	W	6P-12A	SU	<6H	6A-12P	D	CD	NT	C	NDT	
32	32	41-50	M	H	FR	MR	LOW	URBAN	PRIMARY	HOME	W	6A-12P	SU	12-24H	12P-6P	D	AL	NT	E/B	NDT	
33	33	41-50	M	H	DWW	MR	LOW	URBAN	ILITERATE	HOME	W	6A-12P	SU	6-12H	6A-12A	D	CD	P	C	NDT	
34	34	41-50	F	H	H.W	MR	MIDDLE	URBAN	PRIMARY	HOME	A	6P-12A	SU	BD	6A-12A	ND	PD	P	C	DT	PH
35	35	41-50	M	M	DWW	MR	MIDDLE	URBAN	PRIMARY	HOME	A	6A-12P	SU	6-12H	6A-12P	D	AL	P	C	DT	
36	36	21-30	F	H	H.W	MR	LOW	URBAN	PRIMARY	HOME	A	6A-12P	SU	<6H	6A-12A	D	FF	P	E/B	DT	F
37	37	51-60	M	H	DWW	MR	MIDDLE	URBAN	PRIMARY	HOME	A	6P-12A	SU	>24	6A-12P	D	CD	NT	E/B	NDT	
38	38	0-10	F	H	UNE	UM	MIDDLE	RURAL	ILITERATE	HOME	A	12A-6A	AC	<6H	6A-12P	D	A	P	C	NDT	
39	39	10-19	F	H	ST	UM	MIDDLE	RURAL	HIGH	HOME	A	12P-6P	SU	>24	6A-12P	D	FF	P	C	NDT	
40	40	31-40	F	H	H.W	MR	LOW	URBAN	HIGH	HOME	A	12A-6A	SU		12A-6A	ND	PD	NT	C	NDT	
41	41	41-50	M	H	PR	MR	MIDDLE	URBAN	PRIMARY	HOME	A	12P-6P	SU	BD	UNK	D	A	P	E/B	DT	EA
42	42	51-60	M	M	JOB	MR	MIDDLE	URBAN	GRADUATE	W.P	A	12P-6P	AC	6-12H	6A-12A	D	A	NT	C	DT	FMA
43	43	41-50	M	H	ST	MR	MIDDLE	URBAN	HIGH	HOME	A	12P-6P	SU	>24	12P-6P	D	FF	NT	C	NDT	

Sl. No.	SUBJECT ID	AGE DISTRIBUTION (years)	SEX	RELIGION	OCCUPATION	MARITAL STATUS	SOCIO-ECONOMIC STATUS	DOMICILIARY	EDUCATIONAL STATUS:	PLACE	SEASON	TIME OF INCIDENT	MANNER	PERIOD OF SURVIVAL	TIME OF DEATH	TREATMENT DONE	REASON	PECULIAR SMELL	GASTRIC MUCOSA	FSL DEDECTED	POISONS
44	44	10-19	F	H	DWW	UM	LOW	URBAN	HIGH	HOME	A	6A-12P	AC	>24	6A-12P	ND	A	NT	C	NDT	
45	45	31-40	M	H	DWW	MR	MIDDLE	URBAN	PRIMARY	HOME	R	6P-12A	SU	BD	UNK	ND	AL	NT	C	NDT	
46	46	21-30	M	H	PR	UM	LOW	URBAN	GRADUATE	OUTSIDE	R	12P-6P	SU	BD	UNK	ND	FF	P	C	NDT	
47	47	21-30	M	H	DWW	MR	LOW	RURAL	PRIMARY	HOME	S	6P-12A	SU	BD	UNK	D	PD	P	C	DT	EA
48	48	41-50	M	H	DWW	MR	LOW	RURAL	ILITERATE	OUTSIDE	R	12P-6P	SU	>24	6A-12A	D	D	P	C	DT	OPC
49	49	31-40	M	H	DWW	MR	LOW	URBAN	ILITERATE	HOME	R	6P-12A	SU	12-24H	6A-12A	D	PD	NT	C	NDT	
50	50	41-50	M	H	DWW	MR	LOW	URBAN	ILITERATE	HOME	R	6P-12A	SU	>24	6A-12A	D	FF	NT	C	DT	EA
51	51	21-30	F	H	H.W	MR	LOW	RURAL	ILITERATE	HOME	R	6P-12A	SU	>24	6A-12A	D	CD	P	C	NDT	
52	52	41-50	M	H	DWW	MR	LOW	RURAL	ILITERATE	W.P	R	6A-12P	SU	6-12H	6A-12P	D	PD	P	C	DT	OPC
53	53	41-50	M	H	DWW	MR	LOW	URBAN	PRIMARY	HOME	R	6P-12A	SU	SD	UNK	ND	PD	P	C	DT	F
54	54	31-40	M	H	DWW	MR	LOW	URBAN	PRIMARY	HOME	R	6P-12A	SU	BD	UNK	ND	PD	NT	C	NDT	
55	55	21-30	M	H	FR	MR	LOW	URBAN	ILITERATE	HOME	R	6P-12A	SU	6-12H	6A-12A	D	FF	P	C	NDT	
56	56	31-40	M	H	OT	MR	LOW	URBAN	PRIMARY	OUTSIDE	S	12P-6P	SU	6-12H	6A-12P	D	NK	NT	C	NDT	
57	57	31-40	F	H	H.W	MR	MIDDLE	RURAL	PRIMARY	HOME	S	6P-12A	SU	>24	6A-12P	D	PD	P	C	NDT	
58	58	41-50	M	H	OT	MR	LOW	URBAN	ILITERATE	HOME	S	6P-12A	SU	>24	12P-6P	D	D	NT	E/B	NDT	FMA
59	59	41-50	M	C	PR	MR	MIDDLE	URBAN	GRADUATE	HOME	S	12P-6P	SU	BD	UNK	ND	FF	P	C	NDT	OPC
60	60	51-60	M	H	UNE	MR	LOW	URBAN	PRIMARY	OUTSIDE	S	6A-12P	SU	>24	12P-6P	D	D	NT	E/B	DT	
61	61	0-10	F	H	H.W	UM	MIDDLE	URBAN	ILITERATE	HOME	S	6P-12A	AC	<6H	12A-6A	D	A	P	C	NDT	
62	62	41-50	F	H	H.W	MR	LOW	URBAN	PRIMARY	HOME	S	6A-12P	SU	6-12H	12A-6A	D	PD	NT	C	NDT	
63	63	41-50	M	H	OT	MR	LOW	RURAL	HIGH	HOME	S	12P-6P	SU	6-12H	6A-12A	D	FF	P	C	NDT	
64	64	10-19	M	H	ST	UM	MIDDLE	URBAN	PRIMARY	HOME	S	6P-12A	AC	6-12H	12A-6A	D	A	NT	C	NDT	
65	65	31-40	M	C	DWW	MR	MIDDLE	URBAN	PRIMARY	HOME	S	12P-6P	SU	6-12H	12P-6P	D	D	NT	C	DT	F
66	66	31-40	M	H	OT	MR	LOW	RURAL	ILITERATE	OUTSIDE	S	6P-12A	SU	BD	UNK	ND	FF	P	C	DT	

Sl. No.	SUBJECT ID	AGE DISTRIBUTION (years)	SEX	RELIGION	OCCUPATION	MARITAL STATUS	SOCIO-ECONOMIC STATUS	DOMICILIARY	EDUCATIONAL STATUS:	PLACE	SEASON	TIME OF INCIDENT	MANNER	PERIOD OF SURVIVAL	TIME OF DEATH	TREATMENT DONE	REASON	PECULIAR SMELL	GASTRIC MUCOSA	FSL DEDECTED	POISONS
67	67	41-50	F	H	H.W	MR	LOW	URBAN	HIGH	HOME	W	6P-12A	SU	6-12H	6A-12P	D	O	NT	C	DT	
68	68	41-50	M	H	DWW	MR	LOW	URBAN	PRIMARY	HOME	W	6P-12A	SU	BD	UNK	ND	O	NT	C	DT	
69	69	41-50	M	H	DWW	MR	LOW	URBAN	PRIMARY	HOME	W	12P-6P	SU	<6H	6A-12P	D	CD	P	C	NDT	OPC
70	70	31-40	F	H	H.W	MR	LOW	RURAL	ILITERATE	OUTSIDE	W	UNK	SU	SD	UNK	ND	FF	NT	C	NDT	OPC
71	71	21-30	F	H	H.W	MR	LOW	URBAN	PRIMARY	HOME	A	6A-12P	SU	<6H	6A-12P	D	AL	P	C	NDT	M
72	72	10-19	F	C	UNE	UM	LOW	URBAN	PRIMARY	HOME	W	6A-12P	SU	BD	UNK	ND	FF	P	C	NDT	
73	73	31-40	M	H	JOB	MR	MIDDLE	URBAN	HIGH	HOME	A	6P-12A	SU	>24	6A-12A	D	D	NT	C	DT	OPC
74	74	41-50	F	H	DWW	MR	LOW	URBAN	ILITERATE	HOME	A	6A-12P	SU	BD	UNK	ND	NK	NT	C	NDT	
75	75	41-50	M	H	JOB	MR	LOW	URBAN	PRIMARY	W.P	A	6P-12A	SU	<6H	6A-12A	D	FF	P	C	NDT	
76	76	31-40	M	H	DWW	MR	LOW	RURAL	ILITERATE	HOME	A	12P-6P	SU	>24	12P-6P	D	NK	P	C	DT	OPC
77	77	21-30	F	H	H.W	MR	MIDDLE	URBAN	GRADUATE	HOME	A	6P-12A	SU	>24	6A-12P	D	PD	NT	C	NDT	
78	78	21-30	M	C	UNE	MR	LOW	URBAN	PRIMARY	OUTSIDE	A	UNK	SU	SD	UNK	ND	O	P	C	DT	OPC
79	79	31-40	F	H	DWW	MR	LOW	URBAN	ILITERATE	HOME	A	12A-6A	SU	>24	12P-6P	D	NK	NT	C	NDT	
80	80	0-10	M	H	UNE	UM	LOW	URBAN	ILITERATE	HOME	A	6A-12P	AC	>24	12A-6A	D	A	NT	C	NDT	
81	81	21-30	M	H	DWW	MR	LOW	RURAL	ILITERATE	HOME	A	12P-6P	SU	>24	12P-6P	D	FF	NT	C	DT	OPC
82	82	61-70	M	C	UNE	MR	MIDDLE	RURAL	HIGH	HOME	A	6A-12P	AC	>24	12A-6A	D	A	NT	E/B	NDT	
83	83	10-19	M	H	DWW	MR	LOW	URBAN	ILITERATE	HOME	A	6A-12P	SU	>24	6A-12A	D	NK	NT	C	NDT	
84	84	41-50	M	H	FR	MR	LOW	URBAN	PRIMARY	HOME	A	UNK	SU	BD	UNK	ND	NK	NT	C	NDT	
85	85	41-50	M	H	DWW	MR	LOW	URBAN	PRIMARY	HOME	A	6P-12A	SU	<6H	12A-6A	D	FF	P	C	NDT	
86	86	31-40	M	H	OT	MR	LOW	URBAN	PRIMARY	OUTSIDE	A	12P-6P	SU	SD	UNK	ND	PD	P	C	NDT	
87	87	31-40	M	H	FR	MR	LOW	URBAN	PRIMARY	HOME	A	6A-12P	SU	6-12H	12P-6P	D	AL	P	C	DT	ZP
88	88	31-40	M	H	DWW	MR	LOW	URBAN	ILITERATE	HOME	A	6A-12P	SU	12-24H	12A-6A	D	PD	P	C	NDT	
89	89	21-30	F	H	H.W	MR	LOW	URBAN	PRIMARY	HOME	A	6A-12P	SU	>24	12P-6P	D	PD	NT	C	NDT	

Sl. No.	SUBJECT ID	AGE DISTRIBUTION (years)	SEX	RELIGION	OCCUPATION	MARITAL STATUS	SOCIO-ECONOMIC STATUS	DOMICILIARY	EDUCATIONAL STATUS:	PLACE	SEASON	TIME OF INCIDENT	MANNER	PERIOD OF SURVIVAL	TIME OF DEATH	TREATMENT DONE	REASON	PECULIAR SMELL	GASTRIC MUCOSA	FSL DEDECTED	POISONS
90	90	21-30	M	H	DWW	MR	LOW	RURAL	ILITERATE	HOME	A	6P-12A	SU	6-12H	6A-12P	D	FF	NT	C	NDT	
91	91	21-30	M	H	DWW	MR	LOW	URBAN	ILITERATE	HOME	A	6P-12A	SU	<6H	6A-12A	D	PD	NT	C	NDT	
92	92	31-40	M	H	OT	MR	MIDDLE	URBAN	ILITERATE	HOME	R	6A-12P	SU	>24	6A-12A	D	PD	NT	C	NDT	
93	93	10-19	F	M	ST	UM	LOW	URBAN	HIGH	HOME	R	6A-12P	SU	>24	6A-12P	ND	NK	P	C	DT	OPC
94	94	10-19	F	H	ST	UM	MIDDLE	URBAN	HIGH	HOME	R	6P-12A	SU	>24	12A-6A	D	CD	NT	C	NDT	
95	95	31-40	M	H	DWW	MR	LOW	RURAL	ILITERATE	HOME	R	UNK	SU	SD	UNK	ND	PD	P	C	DT	OPC
96	96	41-50	M	H	DWW	MR	LOW	URBAN	PRIMARY	HOME	R	6P-12A	SU	12-24H	12P-6P	ND	PD	P	C	NDT	
97	97	21-30	M	H	FR	UM	MIDDLE	URBAN	PRIMARY	HOME	R	6P-12A	SU	6-12H	6A-12P	ND	FF	P	C	NDT	
98	98	21-30	F	H	H.W	MR	LOW	RURAL	PRIMARY	HOME	R	6A-12P	SU	>24	6A-12P	D	NK	P	C	DT	C
99	99	21-30	F	H	H.W	MR	MIDDLE	RURAL	HIGH	HOME	R	12A-6A	SU	>24	12P-6P	D	CD	P	C	NDT	
100	100	31-40	F	H	H.W	MR	LOW	URBAN	PRIMARY	HOME	R	6A-12P	SU	12-24H	12A-6A	D	FF	NT	C	NDT	
101	101	>70	M	H	UNE	MR	MIDDLE	URBAN	ILITERATE	HOME	R	6P-12A	SU	<6H	12A-6A	ND	CD	NT	E/B	DT	FMA
102	102	>70	F	H	H.W	MR	LOW	URBAN	ILITERATE	HOME	R	12A-6A	SU	>24	12P-6P	D	PD	P	C	NDT	
103	103	31-40	M	H	H.W	MR	LOW	URBAN	ILITERATE	HOME	R	6P-12A	SU	>24	6A-12A	D	PD	P	C	DT	OPC
104	104	31-40	F	H	H.W	MR	MIDDLE	URBAN	HIGH	HOME	R	12P-6P	SU	>24	12P-6P	D	FF	P	C	DT	ZP
105	105	51-60	M	H	PR	MR	MIDDLE	RURAL	GRADUATE	HOME	R	6A-12P	SU	BD	UNK	ND	PD	P	C	DT	M
106	106	10-19	F	H	ST	UM	LOW	RURAL	HIGH	HOME	R	12P-6P	SU	12-24H	6A-12P	D	CD	P	C	NDT	
107	107	51-60	M	H	DWW	MR	LOW	URBAN	PRIMARY	HOME	R	12P-6P	SU	BD	UNK	ND	CD	NT	E/B	DT	EA
108	108	41-50	M	H	DWW	MR	LOW	URBAN	ILITERATE	HOME	R	6P-12A	AC	>24	6A-12A	###	A	NT	E/B	NDT	
109	109	21-30	M	H	PR	UM	UPPER	URBAN	GRADUATE	HOME	R	12A-6A	SU	<6H	6A-12P	D	O	P	C	DT	OPC
110	110	41-50	M	H	OT	MR	MIDDLE	URBAN	HIGH	HOME	R	12P-6P	SU	<6H	6A-12A	ND	PD	NT	C	NDT	
111	111	10-19	F	H	ST	UM	LOW	URBAN	HIGH	HOME	R	12P-6P	SU	BD	UNK	ND	O	P	C	NDT	
112	112	41-50	M	C	JOB	MR	MIDDLE	URBAN	ILITERATE	HOME	S	12P-6P	SU	>24	12P-6P	ND	PD	NT	C	NDT	

Sl. No.	SUBJECT ID	AGE DISTRIBUTION (years)	SEX	RELIGION	OCCUPATION	MARITAL STATUS	SOCIO-ECONOMIC STATUS	DOMICILIARY	EDUCATIONAL STATUS:	PLACE	SEASON	TIME OF INCIDENT	MANNER	PERIOD OF SURVIVAL	TIME OF DEATH	TREATMENT DONE	REASON	PECULIAR SMELL	GASTRIC MUCOSA	FSL DEDECTED	POISONS
113	113	31-40	M	H	OT	MR	LOW	URBAN	PRIMARY	OUTSIDE	S	UNK	SU	SD	UNK	ND	D	P	C	DT	OPC
114	114	21-30	F	H	ST	UM	LOW	URBAN	HIGH	OUTSIDE	S	12P-6P	SU	6-12H	6A-12A	D	O	P	C	DT	O
115	115	41-50	M	H	OT	MR	LOW	RURAL	ILITERATE	W.P	S	6P-12A	SU	12-24H	6A-12A	D	CD	NT	C	DT	PDC
116	116	21-30	F	H	H.W	MR	LOW	URBAN	HIGH	HOME	S	12A-6A	SU	6-12H	6A-12A	D	FF	P	C	NDT	
117	117	51-60	M	H	UNE	MR	LOW	RURAL	HIGH	HOME	S	6A-12P	SU	6-12H	12P-6P	D	CD	P	C	DT	C
118	118	21-30	M	H	DWW	MR	LOW	URBAN	ILITERATE	HOME	S	12A-6A	SU	<6H	12A-6A	D	PD	NT	C	DT	C
119	119	41-50	M	C	OT	MR	LOW	URBAN	PRIMARY	OUTSIDE	S	6A-12P	SU	SD	UNK	ND	D	P	C	DT	M
120	120	31-40	F	H	H.W	MR	LOW	URBAN	PRIMARY	HOME	S	6P-12A	SU	BD	UNK	ND	D	P	C	NDT	
121	121	21-30	M	H	OT	MR	LOW	URBAN	ILITERATE	HOME	S	12A-6A	SU	6-12H	6A-12P	D	PD	P	C	DT	M
122	122	21-30	F	H	H.W	MR	LOW	RURAL	PRIMARY	HOME	S	12P-6P	SU	6-12H	6A-12A	D	CD	P	C	NDT	
123	123	31-40	M	H	DWW	MR	LOW	URBAN	ILITERATE	HOME	S	UNK	SU	BD	UNK	ND	PD	P	C	NDT	
124	124	21-30	F	H	H.W	MR	LOW	URBAN	PRIMARY	HOME	S	6P-12A	SU	6-12H	6A-12A	ND	CD	P	C	NDT	
125	125	10-19	M	H	OT	UM	LOW	RURAL	ILITERATE	HOME	S	6A-12P	SU	>24	6A-12P	D	CD	P	C	NDT	
126	126	41-50	M	H	DWW	MR	LOW	URBAN	PRIMARY	W.P	S	6A-12P	SU	>24	12A-6A	D	NK	P	C	DT	OPC
127	127	61-70	M	H	DWW	MR	LOW	URBAN	ILITERATE	HOME	S	12P-6P	SU	6-12H	6A-12A	D	CD	P	C	NDT	
128	128	21-30	F	H	PR	UM	MIDDLE	URBAN	GRADUATE	HOME	S	12A-6A	SU	6-12H	12A-6A	D	CD	NT	C	NDT	
129	129	21-30	M	H	DWW	MR	LOW	RURAL	ILITERATE	HOME	S	6A-12P	SU	>24	6A-12P	D	CD	P	C	DT	OPC
130	130	41-50	M	H	DWW	MR	LOW	RURAL	PRIMARY	HOME	S	12P-6P	SU	>24	12A-6A	D	CD	P	C	DT	OPC
131	131	21-30	F	H	UNE	UM	MIDDLE	URBAN	HIGH	HOME	W	6A-12P	SU	BD	UNK	ND	D	P	C	DT	M
132	132	41-50	M	H	DWW	MR	LOW	URBAN	ILITERATE	W.P	S	6A-12P	AC	BD	UNK	###	A	NT	C	NDT	
133	133	41-50	M	H	DWW	MR	LOW	URBAN	PRIMARY	W.P	S	6P-12A	AC	BD	UNK	ND	A	NT	C	NDT	
134	134	51-60	F	H	H.W	MR	LOW	URBAN	PRIMARY	HOME	W	12P-6P	SU	<6H	6A-12A	D	CD	NT	C	NDT	
135	135	51-60	F	H	H.W	MR	MIDDLE	URBAN	PRIMARY	HOME	W	6A-12P	SU	BD	UNK	ND	D	P	C	DT	M

Sl. No.	SUBJECT ID	AGE DISTRIBUTION (years)	SEX	RELIGION	OCCUPATION	MARITAL STATUS	SOCIO-ECONOMIC STATUS	DOMICILIARY	EDUCATIONAL STATUS:	PLACE	SEASON	TIME OF INCIDENT	MANNER	PERIOD OF SURVIVAL	TIME OF DEATH	TREATMENT DONE	REASON	PECULIAR SMELL	GASTRIC MUCOSA	FSL DEDECTED	POISONS
136	136	10-19	F	H	ST	UM	MIDDLE	RURAL	HIGH	HOME	W	6P-12A	SU	<6H	12A-6A	D	FF	NT	C	NDT	
137	137	41-50	M	H	DWW	MR	LOW	URBAN	PRIMARY	HOME	W	6A-12P	SU	6-12H	12P-6P	D	FF	P	C	DT	OPC
138	138	41-50	M	H	DWW	MR	LOW	URBAN	ILITERATE	HOME	W	6P-12A	SU	>24	12A-6A	D	AL	P	C	DT	F
139	139	31-40	M	H	OT	MR	MIDDLE	URBAN	PRIMARY	HOME	W	12A-6A	AC	<6H	12P-6P	D	A	NT	C	DT	OPC
140	140	21-30	M	H	DWW	UM	LOW	URBAN	PRIMARY	HOME	W	UNK	SU	SD	UNK	ND	AL	P	C	NDT	
141	141	51-60	M	H	JOB	MR	MIDDLE	URBAN	HIGH	HOME	W	6A-12P	SU	BD	UNK	ND	O	P	C	DT	OPC
142	142	51-60	F	H	H.W	MR	MIDDLE	URBAN	PRIMARY	HOME	W	6A-12P	SU	BD	UNK	ND	O	P	C	DT	OPC
143	143	10-19	M	H	DWW	MR	LOW	RURAL	PRIMARY	HOME	W	6P-12A	SU	>24	6A-12P	D	CD	P	C	DT	OPC
144	144	41-50	M	H	DWW	MR	LOW	URBAN	PRIMARY	HOME	W	6A-12P	SU	BD	UNK	ND	AL	P	C	NDT	
145	145	41-50	M	H	DWW	MR	LOW	RURAL	PRIMARY	HOME	W	6A-12P	SU	<6H	12P-6P	D	CD	P	C	DT	OPC
146	146	41-50	M	H	OT	MR	LOW	RURAL	ILITERATE	HOME	W	12P-6P	SU	6-12H	6A-12A	D	CD	P	C	DT	OPC
147	147	31-40	M	H	DWW	MR	LOW	RURAL	ILITERATE	HOME	R	6A-12P	SU	6-12H	6A-12A	D	D	P	C	DT	OPC
148	148	10-19	M	H	ST	UM	LOW	URBAN	HIGH	HOME	A	6A-12P	SU	SD	UNK	ND	CD	P	C	DT	C
149	149	41-50	M	H	PR	MR	MIDDLE	URBAN	GRADUATE	OUTSIDE	A	12P-6P	SU		6A-12P	D	PD	P	C	DT	OPC
150	150	10-19	M	H	ST	UM	LOW	RURAL	HIGH	HOME	A	12A-6A	SU		6A-12P	D	O	NT	C	NDT	
151	151	41-50	M	H	UNE	MR	LOW	URBAN	ILITERATE	HOME	A	12A-6A	SU		6A-12A	D	FF	P	C	NDT	
152	152	41-50	M	H	JOB	MR	MIDDLE	URBAN	GRADUATE	HOME	R	6P-12A	SU	BD	UNK	D	AL	NT	E/B	DT	EA
153	153	41-50	F	H	H.W	MR	LOW	RURAL	PRIMARY	HOME	R	6P-12A	SU		6A-12P	D	D	NT	C	NDT	
154	154	0-10	F	H	UNE	UM	MIDDLE	URBAN	ILITERATE	HOME	R	6A-12P	AC	<6H	12P-6P	D	A	P	C	NDT	
155	155	51-60	M	C	FR	MR	LOW	URBAN	PRIMARY	HOME	R	6P-12A	SU		12P-6P	D	CD	P	C	NDT	
156	156	10-19	F	H	ST	UM	LOW	RURAL	PRIMARY	HOME	R	6P-12A	SU		6A-12P	D	FF	NT	C	NDT	
157	157	51-60	M	H	OT	MR	LOW	URBAN	PRIMARY	HOME	S	6P-12A	SU	SD	UNK	ND	PD	P	C	DT	C
158	158	41-50	M	H	PR	MR	MIDDLE	URBAN	GRADUATE	HOME	S	12P-6P	SU		6A-12A	D	PD	NT	C	NDT	

Sl. No.	SUBJECT ID	AGE DISTRIBUTION (years)	SEX	RELIGION	OCCUPATION	MARITAL STATUS	SOCIO-ECONOMIC STATUS	DOMICILIARY	EDUCATIONAL STATUS:	PLACE	SEASON	TIME OF INCIDENT	MANNER	PERIOD OF SURVIVAL	TIME OF DEATH	TREATMENT DONE	REASON	PECULIAR SMELL	GASTRIC MUCOSA	FSL DEDECTED	POISONS
159	159	10-19	M	H	ST	UM	LOW	URBAN	PRIMARY	HOME	S	6A-12P	SU		6A-12P	D	FF	P	C	NDT	
160	160	41-50	M	H	OT	MR	LOW	RURAL	HIGH	HOME	S	6P-12A	SU	BD	UNK	D	D	P	C	DT	OPC
161	161	41-50	M	H	UNE	MR	LOW	URBAN	ILITERATE	HOME	S	6P-12A	SU		12A-6A	D	AL	NT	E/B	DT	FMA
162	162	31-40	F	C	H.W	MR	LOW	URBAN	HIGH	HOME	S	6P-12A	SU		6A-12P	D	PD	NT	C	NDT	
163	163	31-40	F	H	H.W	MR	LOW	RURAL	PRIMARY	HOME	S	6A-12P	SU	SD	UNK	ND	CD	P	C	DT	M
164	164	10-19	M	H	ST	UM	MIDDLE	URBAN	HIGH	HOME	S	12P-6P	SU		12P-6P	D	FF	P	C	NDT	
165	165	41-50	M	H	DWW	MR	LOW	URBAN	PRIMARY	HOME	A	6P-12A	SU		6A-12P	D	AL	P	C	NDT	
166	166	41-50	M	H	DWW	MR	LOW	URBAN	PRIMARY	OUTSIDE	S	6P-12A	SU		6A-12P	D	D	NT	C	NDT	
167	167	10-19	M	H	ST	UM	LOW	URBAN	HIGH	HOME	S	6P-12A	SU		6A-12P	D	PD	P	C	NDT	
168	168	41-50	M	H	OT	MR	LOW	RURAL	PRIMARY	OUTSIDE	S	6P-12A	SU	BD	UNK	ND	FF	NT	C	NDT	
169	169	31-40	M	C	DWW	MR	LOW	RURAL	PRIMARY	HOME	R	6P-12A	SU	SD	6A-12P	D	PD	P	C	DT	OPC
170	170	21-30	F	H	H.W	MR	MIDDLE	URBAN	HIGH	HOME	R	6A-12P	SU	>24	12A-6A	D	FF	P	C	NDT	
171	171	21-30	F	H	OT	MR	MIDDLE	RURAL	PRIMARY	HOME	R	12P-6P	SU	6-12H	12A-6A	D	CD	P	C	NDT	
172	172	>70	F	H	H.W	MR	LOW	URBAN	ILITERATE	HOME	R	12A-6A	AC	>24	12A-6A	D	A	NT	C	NDT	
173	173	>70	M	H	UNE	MR	LOW	RURAL	ILITERATE	HOME	A	12P-6P	SU	>24	12P-6P	D	PD	NT	C	NDT	
174	174	41-50	F	H	H.W	MR	LOW	URBAN	PRIMARY	HOME	A	6A-12P	SU	>24	12P-6P	ND	PD	NT	E/B	NDT	
175	175	10-19	M	H	DWW	UM	LOW	RURAL	ILITERATE	HOME	A	6P-12A	AC	<6H	12A-6A	D	A	NT	E/B	DT	FMA
176	176	41-50	M	H	DWW	MR	LOW	RURAL	PRIMARY	OUTSIDE	A	6A-12P	SU	6-12H	6A-12A	ND	PD	NT	E/B	NDT	
177	177	41-50	M	H	JOB	MR	MIDDLE	URBAN	HIGH	OUTSIDE	A	6A-12P	SU	<6H	6A-12P	ND	O	NT	E/B	NDT	
178	178	41-50	M	H	OT	MR	MIDDLE	URBAN	HIGH	HOME	A	12P-6P	SU	BD	UNK	ND	CD	NT	C	NDT	
179	179	41-50	M	H	OT	MR	LOW	URBAN	PRIMARY	HOME	A	6A-12P	SU	BD	UNK	ND	AL	P	C	NDT	
180	180	41-50	M	M	DWW	MR	MIDDLE	URBAN	PRIMARY	HOME	W	UNK	SU	SD	UNK	ND	NK	NT	C	NDT	
181	181	31-40	M	H	DWW	MR	LOW	RURAL	ILITERATE	HOME	W	12P-6P	SU	BD	UNK	ND	PD	NT	C	NDT	

Sl. No.	SUBJECT ID	AGE DISTRIBUTION (years)	SEX	RELIGION	OCCUPATION	MARITAL STATUS	SOCIO-ECONOMIC STATUS	DOMICILIARY	EDUCATIONAL STATUS:	PLACE	SEASON	TIME OF INCIDENT	MANNER	PERIOD OF SURVIVAL	TIME OF DEATH	TREATMENT DONE	REASON	PECULIAR SMELL	GASTRIC MUCOSA	FSL DEDECTED	POISONS
182	182	51-60	M	H	DWW	MR	LOW	URBAN	ILITERATE	HOME	W	6P-12A	SU	<6H	6A-12A	ND	AL	P	C	DT	EA
183	183	>70	F	H	H.W	MR	LOW	RURAL	ILITERATE	HOME	W	6P-12A	SU	<6H	12A-6A	D	CD	P	C	NDT	
184	184	51-60	M	H	DWW	MR	LOW	RURAL	PRIMARY	HOME	W	6P-12A	SU	>24	6A-12A	D	O	P	C	DT	M
185	185	41-50	M	H	UNE	UM	LOW	RURAL	ILITERATE	HOME	W	UNK	SU	SD	UNK	ND	NK	P	C	DT	M
186	186	21-30	M	H	UNE	UM	MIDDLE	URBAN	HIGH	OUTSIDE	S	12P-6P	SU	BD	UNK	ND	O	P	C	NDT	
187	187	21-30	M	H	OT	MR	MIDDLE	URBAN	PRIMARY	HOME	S	12P-6P	SU	6-12H	6A-12P	D	FF	NT	C	NDT	
188	188	61-70	F	H	H.W	MR	LOW	RURAL	ILITERATE	HOME	S	6A-12P	SU	6-12H	12P-6P	D	O	NT	C	DT	F
189	189	41-50	M	H	OT	MR	MIDDLE	URBAN	HIGH	OUTSIDE	S	6P-12A	SU	6-12H	6A-12A	D	PD	P	C	DT	EA
190	190	51-60	M	H	DWW	MR	LOW	RURAL	ILITERATE	HOME	S	12A-6A	SU	>24	6A-12A	D	O	NT	C	NDT	
191	191	41-50	F	H	H.W	MR	LOW	URBAN	ILITERATE	HOME	S	12P-6P	SU	>24	6A-12A	D	D	NT	C	NDT	
192	192	41-50	F	H	OT	MR	MIDDLE	URBAN	PRIMARY	HOME	A	6P-12A	SU	SD	UNK	ND	CD	NT	C	NDT	
193	193	41-50	F	H	H.W	MR	MIDDLE	URBAN	HIGH	HOME	R	12P-6P	SU	>24	6A-12A	D	CD	NT	C	NDT	
194	194	10-19	F	H	ST	UM	MIDDLE	URBAN	HIGH	HOME	S	6P-12A	SU	12-24H	6A-12P	D	O	P	C	NDT	
195	195	41-50	F	H	H.W	MR	MIDDLE	URBAN	PRIMARY	HOME	S	12P-6P	SU	BD	UNK	ND	CD	P	C	NDT	
196	196	21-30	M	H	DWW	MR	LOW	URBAN	PRIMARY	HOME	S	6P-12A	SU	>24	6A-12P	D	PD	NT	C	NDT	
197	197	21-30	M	H	DWW	UM	LOW	URBAN	PRIMARY	OUTSIDE	S	12P-6P	SU	>24	12P-6P	D	PD	P	C	NDT	
198	198	21-30	M	H	DWW	MR	LOW	RURAL	HIGH	OUTSIDE	S	6A-12P	SU	>24	6A-12A	D	NK	P	C	DT	EA
199	199	21-30	M	H	DWW	UM	LOW	URBAN	PRIMARY	HOME	S	12P-6P	SU	>24	6A-12A	D	O	NT	C	NDT	
200	200	0-10	F	H	ST	UM	MIDDLE	URBAN	ILITERATE	HOME	W	12P-6P	AC	>24	6A-12A	D	A	NT	C	NDT	
201	201	31-40	F	H	H.W	MR	LOW	RURAL	PRIMARY	HOME	W	6P-12A	SU	<6H	12A-6A	D	FF	NT	E/B	DT	FMA
202	202	31-40	M	H	DWW	MR	LOW	URBAN	PRIMARY	HOME	W	6P-12A	SU	>24	6A-12A	D	PD	NT	C	NDT	
203	203	31-40	M	H	OT	MR	LOW	RURAL	PRIMARY	OUTSIDE	W	12P-6P	SU	12-24H	12P-6P	D	AL	NT	C	NDT	
204	204	51-60	F	H	H.W	MR	MIDDLE	URBAN	PRIMARY	HOME	W	12A-6A	SU	>24	6A-12A	D	CD	NT	E/B	NDT	

Sl. No.	SUBJECT ID	AGE DISTRIBUTION (years)	SEX	RELIGION	OCCUPATION	MARITAL STATUS	SOCIO-ECONOMIC STATUS	DOMICILIARY	EDUCATIONAL STATUS:	PLACE	SEASON	TIME OF INCIDENT	MANNER	PERIOD OF SURVIVAL	TIME OF DEATH	TREATMENT DONE	REASON	PECULIAR SMELL	GASTRIC MUCOSA	FSL DEDECTED	POISONS
205	205	31-40	M	H	DWW	MR	LOW	URBAN	PRIMARY	OUTSIDE	W	UNK	SU	SD	UNK	ND	AL	P	C	DT	M
206	206	31-40	M	H	OT	MR	LOW	RURAL	PRIMARY	HOME	W	6P-12A	SU	>24	6A-12A	ND	CD	P	C	NDT	
207	207	61-70	M	H	UNE	MR	MIDDLE	RURAL	HIGH	HOME	S	12P-6P	SU	>24	6A-12P	D	CD	NT	E/B	NDT	
208	208	10-19	M	H	UNE	UM	LOW	URBAN	HIGH	HOME	S	12P-6P	SU	>24	12P-6P	D	FF	P	C	NDT	
209	209	21-30	M	H	DWW	MR	LOW	RURAL	PRIMARY	HOME	R	UNK	SU	SD	UNK	ND	D	P	C	DT	F
210	210	41-50	M	H	DWW	MR	LOW	URBAN	ILITERATE	HOME	R	6A-12P	SU	BD	UNK	ND	PD	P	C	NDT	
211	211	10-19	M	H	ST	UM	LOW	URBAN	HIGH	HOME	R	12A-6A	SU	BD	UNK	ND	FF	P	C	NDT	
212	212	41-50	M	H	DWW	MR	LOW	URBAN	PRIMARY	HOME	S	12A-6A	SU	>24	6A-12A	D	AL	P	C	DT	OPC
213	213	41-50	F	H	H.W	MR	LOW	RURAL	ILITERATE	OUTSIDE	S	12A-6A	SU	>24	6A-12A	D	CD	NT	E/B	NDT	
214	214	10-19	M	H	ST	UM	LOW	URBAN	HIGH	HOME	S	6A-12P	SU	>24	6A-12A	D	D	NT	C	NDT	
215	215	21-30	F	H	H.W	MR	LOW	URBAN	ILITERATE	HOME	S	UNK	SU	SD	UNK	ND	NK	NT	E/B	NDT	
216	216	41-50	M	H	DWW	MR	MIDDLE	URBAN	GRADUATE	HOME	S	6P-12A	SU	BD	UNK	ND	CD	P	C	DT	OPC
217	217	10-19	M	C	ST	UM	MIDDLE	URBAN	HIGH	HOME	S	6A-12P	SU	12-24H	12A-6A	D	PD	NT	C	NDT	
218	218	0-10	M	H	FR	MR	LOW	URBAN	ILITERATE	OUTSIDE	S	12P-6P	AC	<6H	12P-6P	D	A	NT	C	NDT	
219	219	31-40	F	H	H.W	MR	LOW	URBAN	PRIMARY	HOME	S	UNK	SU	>24	6A-12A	D	CD	P	C	DT	M
220	220	10-19	M	H	ST	UM	MIDDLE	URBAN	HIGH	HOME	S	6P-12A	SU	BD	UNK	ND	PD	P	C	NDT	
221	221	21-30	M	H	H.W	MR	LOW	URBAN	ILITERATE	HOME	R	6A-12P	SU	<6H	6A-12A	D	NK	NT	E/B	NDT	
222	222	41-50	M	C	PR	MR	MIDDLE	URBAN	GRADUATE	HOME	A	6P-12A	SU	BD	UNK	ND	AL	P	C	DT	M
223	223	41-50	M	H	OT	MR	LOW	RURAL	PRIMARY	OUTSIDE	A	UNK	SU	12-24H	12A-6A	D	NK	P	C	DT	M
224	224	41-50	M	H	OT	MR	MIDDLE	URBAN	PRIMARY	HOME	A	UNK	SU	SD	UNK	ND	D	NT	C	NDT	
225	225	31-40	M	H	DWW	MR	LOW	URBAN	ILITERATE	HOME	A	6P-12A	SU	<6H	12P-6P	D	CD	NT	C	NDT	
226	226	10-19	M	H	ST	UM	LOW	URBAN	HIGH	HOME	W	UNK	SU	SD	UNK	ND	D	P	C	DT	OPC
227	227	21-30	F	H	H.W	MR	LOW	RURAL	PRIMARY	HOME	W	6A-12P	SU	<6H	12A-6A	D	CD	NT	C	NDT	

INSTITUTIONAL ETHICAL COMMITTEE
GOVT.KILPAUK MEDICAL COLLEGE,
CHENNAI-10
Protocol ID. No. 15/2015 Dt: 22.12.2015
CERTIFICATE OF APPROVAL

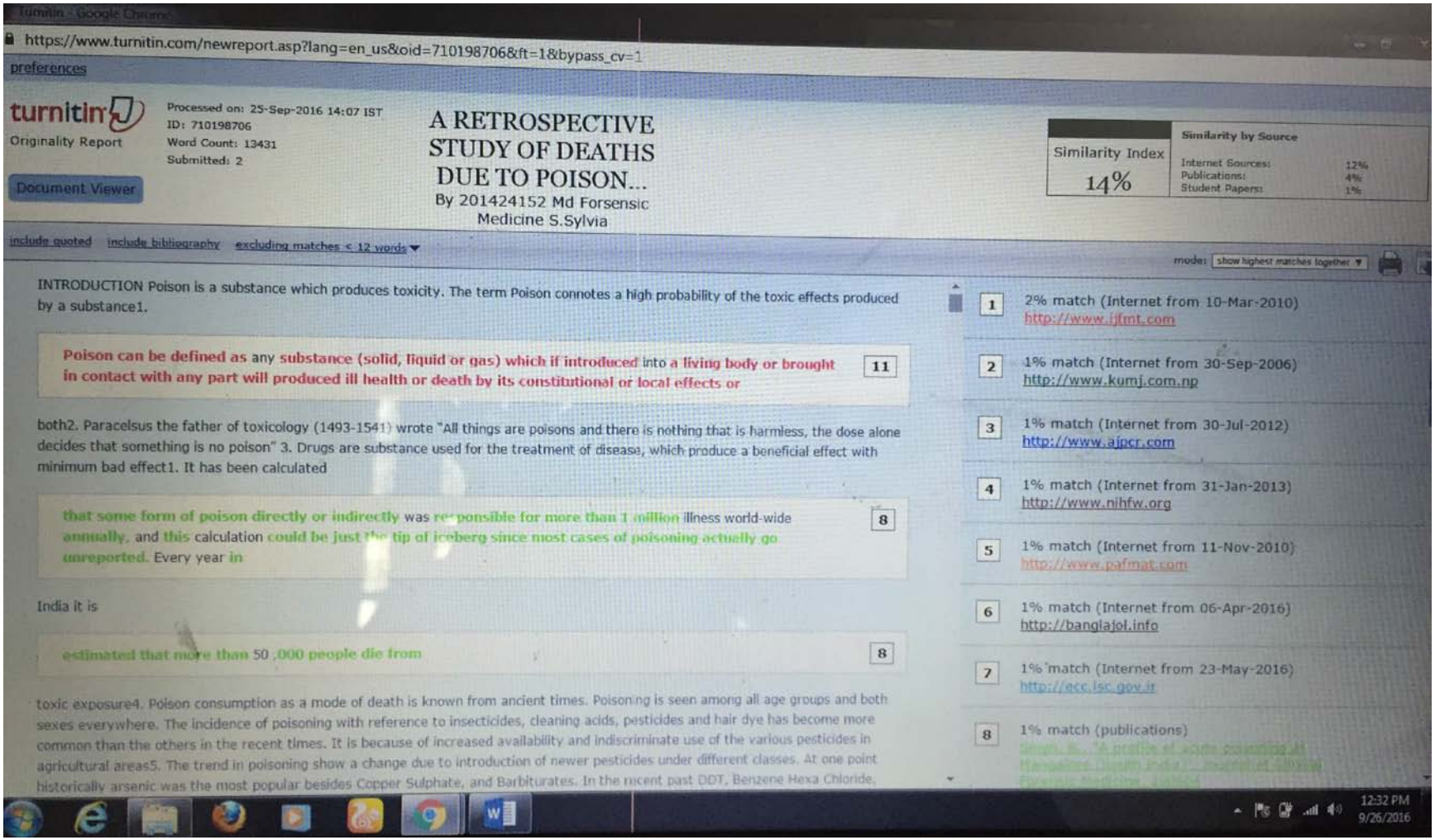
The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "A retrospective study of deaths due to poisoning in Govt. Kilpauk Medical College, Chennai - For Project Work submitted by Dr.S.Sylvia, PG MD Forensic Medicine, Department of Forensic Medicine, Govt. Kilpauk Medical College, Chennai – 10.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

O/C
5/1/16

DEAN,
Govt.Kilpauk Medical College,
Chennai – 10.


Turnitin - Google Chrome
https://www.turnitin.com/newreport.asp?lang=en_us&oid=710198706&ft=1&bypass_cv=1
preferences
turnitin
Originality Report
Processed on: 25-Sep-2016 14:07 IST
ID: 710198706
Word Count: 13431
Submitted: 2
Document Viewer
A RETROSPECTIVE STUDY OF DEATHS DUE TO POISON...
By 201424152 Md Forsensic Medicine S.Sylvia
Similarity Index
14%
Similarity by Source
Internet Sources: 12%
Publications: 4%
Student Papers: 1%
include quoted
include bibliography
excluding matches < 12 words
mode: show highest matches together
INTRODUCTION Poison is a substance which produces toxicity. The term Poison connotes a high probability of the toxic effects produced by a substance1.
Poison can be defined as any substance (solid, liquid or gas) which if introduced into a living body or brought in contact with any part will produced ill health or death by its constitutional or local effects or
11
both2. Paracelsus the father of toxicology (1493-1541) wrote "All things are poisons and there is nothing that is harmless, the dose alone decides that something is no poison" 3. Drugs are substance used for the treatment of disease, which produce a beneficial effect with minimum bad effect1. It has been calculated
that some form of poison directly or indirectly was responsible for more than 1 million illness world-wide annually, and this calculation could be just the tip of iceberg since most cases of poisoning actually go unreported. Every year in
8
India it is
estimated that more than 50 ,000 people die from
8
toxic exposure4. Poison consumption as a mode of death is known from ancient times. Poisoning is seen among all age groups and both sexes everywhere. The incidence of poisoning with reference to insecticides, cleaning acids, pesticides and hair dye has become more common than the others in the recent times. It is because of increased availability and indiscriminate use of the various pesticides in agricultural areas5. The trend in poisoning show a change due to introduction of newer pesticides under different classes. At one point historically arsenic was the most popular besides Copper Sulphate, and Barbiturates. In the nicent past DDT, Benzene Hexa Chloride,
1 2% match (Internet from 10-Mar-2010) http://www.ijfmt.com
2 1% match (Internet from 30-Sep-2006) http://www.kumj.com.np
3 1% match (Internet from 30-Jul-2012) http://www.ajpcr.com
4 1% match (Internet from 31-Jan-2013) http://www.nihfw.org
5 1% match (Internet from 11-Nov-2010) http://www.pafmat.com
6 1% match (Internet from 06-Apr-2016) http://banglajol.info
7 1% match (Internet from 23-May-2016) http://ecc.isc.gov.ir
8 1% match (publications)
12:32 PM
9/26/2016